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LOBAR PNEUMONIA.

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Synonyms.—Croupous pneumonia, Fibrinous pneumonia, Pneumonitis, Lung Fever.

The lung differs from all other structures in having two separate circulations: the nutritive, supplied from the left side of the heart through the bronchial arteries, and the functional, supplied from the right side of the heart through the pulmonary artery. This double circulation underlies all the phenomena of pneumonia, and must be recognized in any definition of the disease, as without it the disease itself could not exist.

Definition.—Lobar pneumonia is an acute disease in which a specific parasite invades the air cells of one or more pulmonary lobes, where it grows in a fibrinous medium exuded from the functional capillaries, and generates a toxin that infects the system at large.

The local process causes consolidation of the affected area by filling the air cells with the effused material, which, material is afterwards removed, leaving the structure of the lung intact.

The general infection is marked by fever, which, in a typical case, begins with a chill, and after a duration of from four to nine days ends abruptly by crisis.

In most cases a local dry pleuritis is excited, the phenomena of which are added to those of the pneumonia proper.

Death may take place from the virulence of the infection, from loss of respiratory surface, from exhaustion of the right heart, from consecutive asthenia, from complications, or from a combination of two or more of these causes.

Symptoms.

A brief sketch of the principal clinical features of the disease will be useful as affording a preliminary view of the field to be examined.

The attack may be preceded by prodromes to be hereafter described, but as a rule the first complaint of the patient is of a pain in the chest, usually in the mammary region. This is often very severe, and by restricting the movements of the ribs renders the respiration superficial and rapid. A chill more or less severe follows,

or in many cases precedes, the pain. The chill varies from a mere creeping sensation to a heavy and prolonged rigor, as severe as in a case of intermittent fever. Coincident with the chill there is a rise of temperature. The thermometer shows from three to four degrees Fahrenheit of fever during the first twelve hours, rapidly rising until the temperature reaches from 103° to 105° F. or even more. Then there is a period during which the temperature is maintained with slight variations until from the fifth to the eighth day, when a crisis occurs and the temperature falls abruptly, until within a few hours it becomes normal.

Cough is an early symptom, but it is repressed as much as possible to avoid the severe pain it causes. The expectoration is apt to be frothy at first, and mixed with florid blood; later it becomes viscid and very tenacious, so that it is spat out with difficulty and adheres like thick mucilage to the vessel containing it. Its color at this stage varies in different cases. It may be a light yellow, a pale green, or a chocolate-brown, or a mixture of these colors. It is often likened to prune juice. Sputa of this character may be considered pathognomonic of this disease. As resolution progresses the expectoration becomes less colored, less sticky in consistence, and more catarrhal in its character, and the quantity gradually diminishes until it ceases altogether.

The respirations are early increased in frequency, and this quite out of proportion to the increase in pulse rate and temperature. With a pulse of not more than 90 the respirations will number 30 or more to the minute. In nearly every severe case there will be a time when the respirations will go up to 40 or 50, and they not infrequently reach 60 or more per minute, when the consolidation is extensive or pulmonary œdema takes place. The pulse is usually full and strong in the early stages, numbering from 90 to 100 when the temperature is 103.5° to 104°, and becoming weaker and more frequent as the disease advances. When the respiration is greatly embarrassed the pulse is apt to be small and creeping, unless affected by stimulants.

The skin is hot and dry at first, later there is a tendency to perspiration which may be profuse. The face is pale, with often a dusky red patch on either cheek. The lips are inclined to a bluish hue in proportion to the degree of pulmonary implication. They are often the seat of an herpetic eruption.

After the first forty-eight hours the chlorides in the urine are greatly diminished in quantity, or entirely absent. In severe cases a moderate degree of albuminuria is common during the height of the fever.

The physical signs begin to be appreciable as a rule within from

twelve to twenty-four hours after the initial chill. Usually the first to be noticed is a fine crepitant râle, heard only with inspiration, though in some cases this is preceded by a diminished clearness of the respiratory murmur. Dulness on percussion succeeds, increasing in intensity as the consolidation becomes more complete. Ultimately the respiratory murmur is wholly replaced in the affected area by a peculiar whiffing sound heard most distinctly towards the close of expiration, the so-called tubular breathing. If the pleura is involved there may be a rubbing or creaking sound in addition. There are increased vocal resonance and vocal fremitus.

As resolution progresses the auscultatory signs reappear and again disappear in the reverse order of their original sequence. At the crisis, while the temperature falls and the pulse and respiration become less frequent, there is no immediate change in the physical signs, showing that the condition of the affected area remains the same.

In a large proportion of cases of pneumonia there is decided leucocytosis, the white cells numbering 20,000, 30,000, 40,000 or more to the cubic millimetre.

This being a general picture of the disease we will examine its features more in detail, noting as we proceed the variations more or less frequently observed from the typical course of the affection.

Prodromes.—In about twenty-five to thirty per cent. of the cases of pneumonia there are prodromes. In the aged the proportion is higher, reaching to sixty per cent.[1] There may be for several days before the seizure a general feeling of malaise, headache, loss of appetite, dull pains in limbs and back, perhaps diarrhœa or epistaxis. The skin may have a sallow appearance suggesting slight jaundice. Chilly sensations alternating with flashes of heat may be experienced.

These symptoms, leading up to the fully developed attack, are accepted as a part of the seizure, an initial stage of the pneumonia depending upon the specific infection.

Andral[2] relates a case in which the general symptoms of headache, debility, dulness of the intellectual faculties, flushed face, injected eyes, and a frequent pulse appeared at least six days before the physical signs could be detected.

The Initial Pain.—This is usually sudden in its onset. Not infrequently the patient is awakened in the night by a sharp pain in the chest, much aggravated by every movement of respiration. This pain may be felt in any part of the thorax, but is most often referred to the region of the nipple on the affected side. Instinctively the respiratory movements are restricted in amplitude, and to compensate for this become more frequent. It will be observed that the

sound side of the chest expands more than its fellow. There are cases, however, in which there is only a dull heavy aching, and sometimes no complaint of pain whatever is made. When the pain is sharp it is no doubt due to accompanying pleuritis, while the dull aching has its seat in the pulmonary parenchyma, the pleura not being involved, and the dull pain not being masked by the more severe.

In some instances the pain is referred to a point entirely outside of the chest. Thus, I recall a case in which the pain, which was very acute, was felt in the abdomen, and attention was thus diverted for a time from the real seat and nature of the disease. In many cases the pain is felt with great intensity some hours or even a day or two before there is any considerable rise in temperature or before the physical signs can be recognized. That the pleura should be thus irritated before there are any other symptoms or signs of pleurisy, and while the pneumonic process is still undeveloped, would seem to indicate a special susceptibility of the serous membrane to the very early local action of the toxin. It has been suggested that the pain was due to the stretching of the inflamed pleura by the swelling of the subjacent lung,[3] but this is at variance with the facts above mentioned.

The duration of the pain does not often exceed two or three days. As consolidation becomes more complete the movements of the lung nearly cease, and the friction of the pleural surfaces upon each other being less, the pain is diminished in proportion.

The Chill.—This is not uniformly present. It was absent in 35 per cent. of 223 cases observed at the Presbyterian Hospital. Elsner found it absent in 30 out of 150 cases.[4] It marks the moment at which the system feels the brunt of the infection, and from what we now know of the infectious diseases as a class, its occurrence alone would raise the presumption that pneumonia was such a disease. It varies greatly both in intensity and duration; in one case scarcely attracting the notice of the patient, and in another occurring as a severe rigor lasting for an hour or more, and leaving after it a sense of profound depression. Its severity generally bears a relation to the severity of the infection as expressed by the effect upon the nervous system rather than upon the temperature, and it has a prognostic significance which will be dwelt upon in another section.

It is sometimes, though not often, repeated when a fresh invasion of lung occurs. Usually the cases in which it is absent are mild, or they have the type of bronchopneumonia rather than of the distinctively croupous form.

The relation of the chill to the local condition is not clear. By

many it is considered as marking a general infection of which the lung changes are a subsequent expression. In proof of this view it is put forward that the physical signs are often not developed until some time has elapsed after the chill. Indeed, Syers[1] reports five cases of pneumonia entirely without physical signs of lung consolidation. In each case the illness began suddenly; the temperature was high and fell by crisis; there was labial herpes; and the pulse-respiration ratio was typically that of pneumonia. Still no evidence of lung consolidation was detected in the physical examinations, which were repeated and most thorough. Now while such cases would seem to support the view that pneumonia is primarily a general infection with a secondary local lesion of varying intensity and importance, or which may remain absent altogether, it seems impossible from a careful consideration of all the phenomena to resist the conviction that the disease *begins* in the lungs.

The evidence to the contrary is based on the general assumption that there is no disease in the lungs unless, and until, there are physical signs indicating its presence. But a moment's reflection will show that as the local disease causes the signs, it must *precede* their appearance, and that it must progress to a certain extent before it can manifest itself in a way to be appreciated through all the intervening tissues by the coarse and insensitive methods of auscultation and percussion. That bacteria are developed to a considerable extent during this time, and are actively engaged in the production of toxin, is *a priori* a reasonable inference from the toxæmia which is already present. If we reject this inference we must fall back upon the assumption that some other form of toxin from a microorganism in some other situation exists first, and in some unexplained way ultimately induces the growth of the specific organism in the lungs. Such an assumption is unreasonable in itself, and requires more for its support than the mere absence for a time of the physical signs. Moreover, we find that when rigors occur in other infective diseases, they often appear before there is any local manifestation at all commensurate with the general disturbance. This is especially true of purulent infection. The chill generally precedes any palpable local change, and its appearance is often the first indication that a pus focus is about to develop. Yet we must believe that the initial step is a local infection which even at its inception, so far as we can distinguish, is competent to poison the general system. Just how much toxic material has to be taken up in order to provoke an explosion we do not know, but there is nothing in analogy to forbid the inference that, in pneumonia, a local process going on in the air cells may furnish enough toxin to excite a general reaction without giving rise as yet

to recognizable physical signs. Besides, under the title of prodromes certain symptoms have been mentioned which in a considerable proportion of the cases *precede the chill*. Granting them to be a part of the disease, we have to admit that there is toxin in the blood at a very early stage, often as long as two or three or more days before the physical signs begin to be discoverable. But if there is toxin in the blood, there must be germs somewhere, and where if not in the lung? Hence the inference already drawn seems to be inevitable.

In this connection it is to be noted that different specimens of pneumococci produce toxins differing enormously in virulence, so that in one case a comparatively limited local action may give rise to an intense general infection, while in another case a much greater accumulation of cocci in the lung may have a mild infection as the result.

The Pulse.—In sthenic cases the pulse is at first full, bounding, and incompressible; such a pulse as in former times was held to demand blood-letting. Its frequency bears about the normal relation to the temperature, but less than the normal relation to the respiration. As the disease progresses the pulse tends to increase in frequency and to become less firm. When the pulmonary obstruction is considerable and the blood current is retarded in the lungs the arterial system is left but partly filled and the pulse becomes small and thready. Under these conditions it is often observed to fluctuate in strength with inspiration and expiration, being feebler when the blood is aspirated towards the thorax by the expansion of the chest, and stronger when the contraction of the latter is added to the force of the cardiac systole.

As a rule the pulse decreases in frequency with the fall of the temperature at the crisis. But this is not always so, and a frequent pulse with a low temperature is an association of serious import. It indicates that the system has become deeply infected, and the heart muscle proportionately weakened. In an adult a pulse as high as 120, if maintained from day to day, may well cause serious apprehension. On the other hand, especially in elderly persons, there may be little or no increase in the frequency of the pulse. In young children it often reaches a very high figure, which may even be difficult to count, and yet the danger is not proportionately greater. In fact the pulse is subject to so great variations in sympathy with the disturbance of the nervous system that its indications taken alone are extremely unreliable.

The *respiration* early assumes a frequency which is very characteristic, and is sufficient of itself to excite a suspicion of the nature

of the disease. This increased frequency is due to several causes. Probably there is from the very beginning a specific irritation which is felt in the affected territory in the lung and which excites to greater functional activity.

Hughlings Jackson[5] mentions a case in which the intercostal muscles acted in voluntary, but not in involuntary, respiration. This was one of several cases of pneumonia in which the knee-jerk was absent.

Such cases serve to show the profound effect which the toxin is capable of exerting upon the nervous system, and which extending to the respiratory centre would suggest a partial explanation of the great rapidity of respiration while as yet the respiratory surface is scarcely diminished. In confirmation of this, Washbourn found that mice injected with Fränkel's pneumococcus suffered from dyspnœa even in the absence of pneumonia.

Next, the pain which is felt in most cases tends to prevent a full expansion of the thorax, and there is consequently an effort to make up by frequency of breathing what is lost in amplitude. This is evidenced by the superficial character of the respiration, and the fact that even before the expansion of the lung is lessened by solidification the affected side of the chest may often be seen to move less freely than the other. Then comes the influence of the fever, which of itself should give about one additional respiration for each increase of four beats of the pulse, or one degree Fahrenheit of temperature. Lastly there is added, as congestion and consolidation become factors in the case, the effect of reduced respiratory surface.

These causes combined result in a frequency of the respiration out of all proportion to the pulse and temperature. Thus seventeen cases of pneumonia observed with reference to this point at the Presbyterian Hospital gave as the average maximum of temperature, pulse, and respiration, 104.4°, 131, and 57 respectively. It will be noticed that the usual ratio of the pulse to the temperature, viz., increase of ten beats for each degree of rise, is very closely maintained, while the usual ratio of one respiration to four pulse beats is changed to about one to two and two-thirds.

At the crisis the respiration falls with the pulse and temperature, but not in equal degree, running usually between 20 and 25, while the temperature is normal or subnormal, and the pulse about 80. This fall of the respiration without a corresponding change in the available breathing surface is often cited as a proof that the pulmonary conditions have very little share in making up the gravity of the case. But, it is not to be overlooked that after the crisis the respiration maintains its comparatively low rate only in the

absence of all muscular effort, and when there is no accession of temperature. Should the patient attempt any exertion, or should the temperature rise again from any cause an immediate quickening of the respiration to a disproportionate degree would show the crippled condition of the lung. There is normally a wide margin of respiratory surface which can be drawn upon without greatly disturbing the breathing. But, with this margin exhausted the slightest tax upon the respiration either by muscular effort or by rise of temperature makes itself felt at once.

Pregnancy, rachitis, Pott's disease, pulmonary emphysema, and all conditions that interfere mechanically with the movements of respiration, add to the dyspnœa, and must be taken into consideration in estimating the degree to which the lungs are affected by the pneumonia itself.

Grisolle insists upon the distinction between real and apparent dyspnœa in pneumonia. The nervous patient, tormented by the stitch that limits the excursion of the ribs, and by an incessant cough will breathe 75 to 80 times per minute. Yet he is really less asphyxiated than the prostrated cyanotic patient who breathes only 24 to 28 times in the same period. The prognosis is determined more by the peripheral cyanosis, the distention of the jugulars, and the state of the pulse and heart than by the apparent dyspnœa.[7]

In pneumonia in the aged, which, however, is apt not to be strictly lobar or croupous, this frequency of respiration together with a moderate degree of fever is often almost the only evidence of the serious character of the attack, and is sufficient to establish the diagnosis in advance of the appearance of the physical signs, which latter are often delayed or imperfectly manifested.

Very frequently the respiration is interspersed with grunting sounds at intervals or perhaps with every expiration. This is most marked during the stage when the pleurisy is at its height, and is usually associated with a more or less soporous condition, the sound ceasing when the patient is aroused.

In cases in which the respiration is labored the alæ of the nose will be observed to expand with each inspiration. This is an automatic movement, and indicates that the respiratory centre is receiving an unusual stimulus. As a prognostic sign it has no special value.

The *temperature* begins to rise from the moment of the attack, and increases with slight fluctuations until the maximum is reached. In cases that pass the crisis the highest point is usually a few hours before the decided fall takes place, the temperature often declining a degree or so, to rise again before the final drop. When death occurs

before the crisis the highest point often immediately precedes dissolution, when the thermometer may indicate 107°, 108°, or even 109°.

Very high temperatures, however, may be recovered from. Francis Hawkins[1] gives a case of hyperpyrexia with double lobar pneumonia, in which, upon the fifth day of the disease, the temperature rose to 108.4° F. (42.4° C.) as recorded by two different thermometers. The temperature fell by lysis, and became normal on the twenty-ninth day after admission. Patient discharged recovered. The treatment was cold sponging and packing with iced water.

When defervescence occurs by crisis, which is usually from the fifth to the eighth day, the temperature falls within a few hours almost or quite to normal. This is apt to occur during the night, and it often happens that the patient is left at the evening visit with no sign of an approaching decline in temperature, yet the next morning is found in an almost afebrile condition.

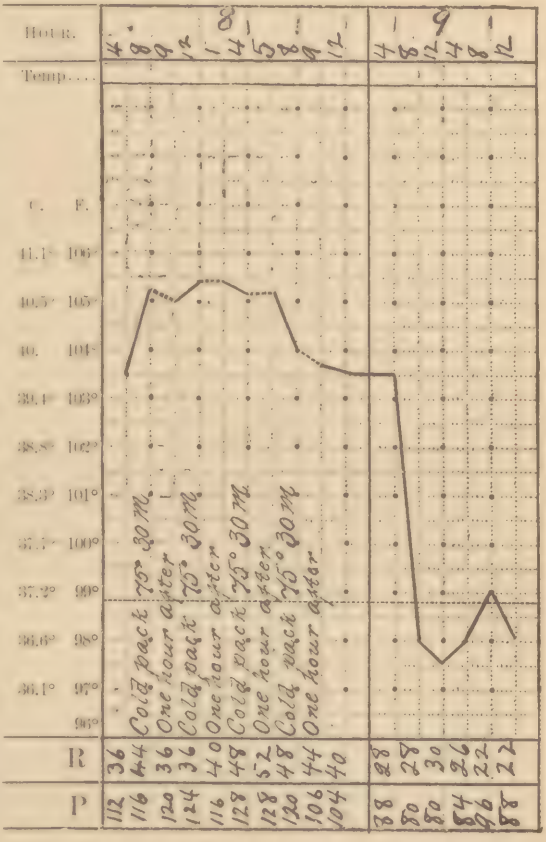

Chart No. 1.—Defervescence by Crisis.

The accompanying chart (No. 1) is of a case admitted on the eighth day of the disease. Pack at 75° F. repeated every four hours produced very little effect, but at the crisis on the ninth day the temperature fell 5½° F. in four hours.

A day or two before the actual crisis there may occur a sudden and considerable drop of temperature, followed promptly by a rise to its former height. This is known as pseudo-crisis. On the other hand, the crisis may be preceded by a brief but marked accession of temperature, the so-called critical perturbation.

An increase of temperature following a decline suggests the involvement of a fresh portion of lung, though it may be due to other causes.

As will be seen later under the head of Prognosis, the severity of the attack is not necessarily in proportion to the temperature; indeed, the greatest danger may be present when the thermometer shows but a moderate rise. Especially in the aged, it is possible for the attack to be afebrile throughout, and to go on to a fatal termination without rise of temperature.

It is to be borne in mind that the temperature of the surface may be but little increased, while that of the interior of the body is alarmingly high. Hence the rectal temperature may be greatly in excess of that observed in the axilla or in the mouth. This is not seldom the case in elderly persons, in whom the peripheral circulation is sluggish. It is also occasionally the result of extreme intensity of the initial infection. Especially in children this high internal temperature may coincide with coldness of the extremities.

At the crisis the face becomes paler and calmer, restlessness and delirium disappear, the skin becomes moist, or there may be profuse sweating, and there is a tendency to sleep. An attack of diarrhœa at this time is not rare. The temperature may fall in from twelve to eighteen hours to 98° or 97° F., but may rise again on the following day to 99° or 100°.

Fowler gives the following as the days of crisis in the order of their frequency: "22 per cent. on the seventh day; 16 per cent. on the fifth day; 12 per cent. each on the sixth and eighth days; and 10 per cent. on the ninth day."

Instead of a complete crisis there may be a considerable and rapid decline of temperature, followed by a rise, and this succeeded in turn by a gradual lysis. Again, there may be a complete well-marked crisis, but the lung remains solid; there is renewed pyrexia and the case goes on to a fatal termination.

In a considerable proportion of cases there is no such sudden fall of temperature as constitutes a crisis, but the temperature, while fluctuating more or less, gradually becomes lower until the normal line is reached. This defervescence by lysis may be complete at any time between the third and the fifteenth or twentieth day (see chart No. 2).

The *cough* is seldom a marked feature. It is accompanied by severe pain in most cases, due to accompanying pleuritis, and for this reason is superficial and catchy. Usually it begins within the first few hours, or in some instances it precedes the distinctive symptoms of the attack. Sometimes, particularly in persons past middle life, it

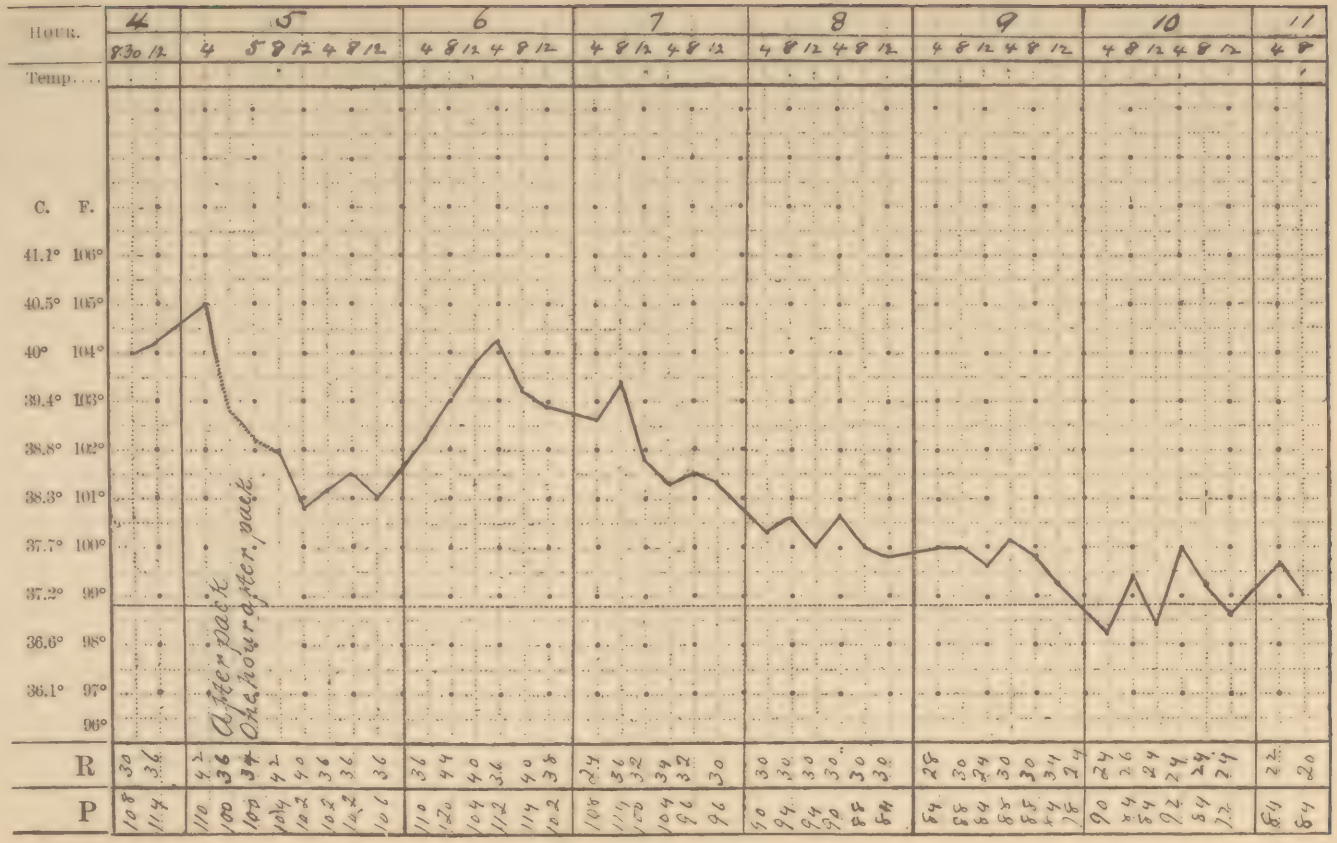

CHART No. 2.—Defervescence by Lysis.

is entirely absent. Severe cough occurring in paroxysms shows that the bronchial surface is involved. Such a cough is usually limited to the early stage of the attack. While it lasts it is the source of considerable distress from the pleuritic pain excited by it.

The *expectoration* is at first frothy and tinged with blood. Occasionally it is almost pure blood. Later it becomes more dense and takes on a yellow, or brownish, or rusty color—according to the amount of blood it contains. The color is not often uniform, but more or less marbled from the aggregation of differently stained masses, varying in color from dark reddish-brown to pinkish or amber, or a greenish tint. With all these colors there is, however, a vitreous, semitranslucent appearance, which is not lost until the abundant admixture of leucocytes at a later stage renders the expectoration opaque. The quantity is not great, seldom exceeding two or three ounces in twenty-four hours. The expectorated material is composed of mucus mixed with leucocytes, red blood cells, epithelia (both columnar and pavement), fibrinous casts of the air cells, formerly described as pneumonic globules, and occasional casts of the bronchioles. Curschmann's spirals are sometimes found in the sputum, but their presence has no diagnostic significance.

The sputum abounds in microorganisms, especially the diplococcus lanceolatus, which, however, diminish rapidly after the crisis. Chemically, it is found to contain a large amount of sodium chloride, while the alkaline phosphates are absent. The extreme adhesiveness of the expectoration has been already referred to. Owing to this it is difficult to eject it from the mouth, as it adheres to the tongue and teeth, often requiring the aid of a handkerchief for its removal.

Later it assumes a more creamy appearance and becomes somewhat less tenacious. At this stage it contains a larger proportion of leucocytes, many of them in a state of fatty degeneration.

Occasionally, instead of this thick sticky material we find a thin fluid deeply stained with a dark coloring-matter derived from the blood. This so-called prune-juice expectoration is supposed to denote a specially unfavorable condition. Probably it depends upon a serous exudation into the air passages, and indicates that the blood is less rich in fibrin than in cases yielding the tenacious sputum.

Now and then we meet with a case of pneumonia in which there is no expectoration from beginning to end. In these cases the exudate is removed by absorption alone. The process of freeing the lung and restoring its permeability is not materially longer than when a part of the effused material is coughed up. Indeed, when we consider the large amount of material to be removed, it is evident that the quantity represented by the expectoration must count for very

little in the process. In fact, the bulk of the expectorated material in any case is furnished by the coincident bronchitis.

An expectoration such as is described above is fairly pathognomonic. In no other affection do we have these sticky, tenacious, variously colored sputa. In bronchitis occasional isolated masses somewhat resembling them in character may be found, intermixed with watery and frothy material, but they lack the adhesive quality by which the whole contents of the cup are bound together, and they assume rather more of a nummular form.

The *posture* in pneumonia is indicative of a chest affection. The patient lies at first on the back, as rendering the respiration less painful; but as consolidation progresses, the increased weight of the affected lung makes the decubitus on that side more comfortable than any other position, as there is less pressure upon the sound lung, and the breathing is therefore easier. There is not the frequent change of posture so common in simple pleurisy; the patient having found the position that suits him the best, is likely to maintain it pretty steadily. Orthopnœa is rarely observed, unless there is fluid in the pleural cavity or a considerable degree of pulmonary œdema.

The *face* is apt to have a more or less dusky hue, in proportion to the respiratory involvement, and the lips are pale with a bluish tint in most of the severe cases. The countenance has a pained and anxious expression and the aspect is that of a person seriously ill. There is often evidence of great weakness and a tendency to slip down in the bed, which may be quite as marked as in typhoid fever. This, however, is far from being a constant feature, some persons retaining a remarkable degree of strength throughout the whole attack. The difference in this respect will be referred to again in another connection.

The Blood.—From the outset the number of leucocytes in the blood is usually considerably increased, reaching sometimes as high as 40,000 to 60,000 per cubic millimetre. According to Stienon,[9] during the pyrexia the polynuclear cells are very abundant and the eosinophiles very rare. After the crisis the former diminish, and the latter increase. The subject of leucocytosis will be further considered under the head of Prognosis.

It has long been known that in pneumonia the proportion of fibrin in the blood is greatly increased. This increase may amount to as much as two hundred and fifty per cent.

The Urine.—The absence of chlorides from the urine is a pretty constant condition after the first two or three days. The quantity becomes less as the process of consolidation advances, and, as the

exudate in the lung is enormously rich in chlorides, it seems a fair inference that they escape into the alveoli in place of being excreted by the kidneys. This inference is strengthened by the fact that during the absorption of the exudate the quantity of chlorides thrown out by the kidneys is abnormally great. In obscure cases this disappearance, partial or complete, of the chlorides from the urine may have considerable diagnostic value, although it may occur in other diseases.

F. Pick [10] calls attention to a change in the urine in pneumonia not described hitherto. In from twenty-four to forty-eight hours after the crisis there is a decided decrease of acidity, the urine becoming neutral or alkaline. This condition continues for from twenty-four to thirty-six hours and then the acidity returns.

The phenomenon is of pretty constant occurrence, Pick having observed it in thirty-one out of thirty-eight cases. He ascribes it to the absorption of the exudate, which is rich in sodium. It may be due, however, to sodium being excreted in the urine as bicarbonate, which under normal conditions would have been neutralized by pneumic acid in passing through the lungs (see page 44).

In a considerable proportion of cases of pneumonia albumin is found in the urine. Owen found it in all but four of twenty-six cases.[11] The amount bears some relation to the severity of the case. The albuminuria is probably the result of several contributing causes. The infection itself, like other acute infections, has a tendency to produce this result, the pneumotoxin probably irritating the kidneys as so many other toxins do. Secondly, the interference with the pulmonary circulation results in more or less tension of the venous system, congesting the kidneys; and lastly, the imperfect metabolism consequent upon incomplete hæmatosis leaves material in the blood to be thrown out by the kidneys, which material normally would be destroyed by oxidation.

Besides albumin, blood and fibrinous casts of the uriniferous tubes are occasionally found in the urine.

According to Sternberg,[12] secondary infection of the kidneys by the micrococcus pneumoniæ crouposæ is probably not infrequent. The micrococcus is not ordinarily found in the blood in pneumonia, though sometimes present in small numbers. Fraenkel and Reiche (1894) found this micrococcus in the kidneys in twenty-two out of twenty-four autopsies upon cases of pneumonia, and had a bacteriological examination of the urine been made during life, it is probable that the pneumococcus would have been found there.

In a certain number of cases, undoubtedly, the kidney disease is due to the presence of the pneumococcus itself in the kidney. Such

a condition could be demonstrated during life only by finding the organism in the urine in a case presenting symptoms of renal irritation.

The most recent observations go to show that cases in which secondary infection by the coccus itself occurs in organs other than the lungs usually prove fatal, and it is probable that these secondary infections very often determine the issue of the primary disease. The albuminuria in the simple form begins to clear up after defervescence, except in cases in which the respiratory embarrassment continues after the fall of the temperature.

Associated with the albumin hæmoglobin may appear in the urine. Nash [14] mentions a case in a girl of sixteen ill four days with pneumonia. The crisis occurred on the seventh day, after which no more coloring-matter or albumin could be detected.

An early appearance of albumin in the urine, before the pulmonary circulation is materially obstructed, may be interpreted as indicating a high degree of infection, and therefore as likely to be followed by severe depression. Usually its first appearance is when the disease is at its height.

The prognostic significance of albuminuria in this affection is important in proportion to the degree of renal implication present. This is judged of by the quantity of albumin on the one hand and the associated symptoms on the other. A large amount of albumin in a scanty urine containing epithelial casts and perhaps blood discs, the fever at the same time being high, the stomach irritable, and the mental faculties clouded, creates a presumption that organic change is present in the kidney. Such change alone constitutes a grave element in the prognosis, but when we consider that lying back of it there is likely to be an infection of the kidney by the organism itself as well as by the toxin, and that in this case the germ being in the blood, other organs also will be involved, it gives an extremely serious aspect to the case. Should an examination of the blood show the presence of active pneumococci, the prognosis would be almost hopeless. Sturges and Coupland found that of twenty-seven cases in which albuminuria of all grades was present five were fatal, while of seventy-one without albuminuria all but two recovered. From these figures it would seem that pneumonia with albuminuria is more than six times as fatal as without it. It would be wrong, however, to infer that such an increase of mortality is due to the kidney affection by itself. We should rather note the fact that this affection is prone to appear in cases that are otherwise bad, and more likely, therefore, to prove fatal.

Regarded as a complication, acute parenchymatous nephritis is

not uncommon. Osler[14] found marked interstitial changes in the kidneys in twenty-five per cent. of the cases that came to autopsy at the Montreal General Hospital. It is very rare that it is the foundation of permanent disease.

A further effect of the disease upon the renal function is a remarkable diminution of the toxicity of the urine. According to researches by Roger and Gaume,[15] the toxicity is only one-fourth of the normal during the pyrexia, but rises to the normal or beyond it at the crisis.

It would seem from this that either the disease so acts upon the chemical processes that the usual amount of toxins is not formed, or that the kidneys fail to eliminate the toxins in full measure from the blood. While it is not at all impossible that the latter is the case in some degree, and that the retained toxins may play a part in producing the general condition, still as the lessened toxicity is not necessarily accompanied by evidence of kidney implication it seems more probable that it depends upon a fault in the chemical processes by which the toxic material is formed.

Throughout the febrile stage the quantity of urea is abnormally great, the urine is scanty, and the specific gravity is excessive.

After the crisis the urine becomes more abundant, but the excess of urea persists for some time, and may be greater even than when the pyrexia was present. This is probably due to the formation of urea from the exudate absorbed from the lung. According to Herter, the uric acid also is greatly increased.

Delirium.—The mental condition is more or less disturbed in a majority of cases. As the pyrexia increases delirium is pretty frequently observed. It may take the form of simple incoherence of speech, or it may be of the active, busy sort. Occurring early and in persons who naturally show a tendency to cerebral derangement in the presence of fever, it may have but little significance. Even the moderate pyrexia attending a severe cold will sometimes cause a passing delirium in such patients, and if attacked with pneumonia they are likely from the outset to appear more ill than they really are, so far at least as the mental condition indicates. But in other cases the delirium is a part of the general nervous perturbation to which the infection gives rise, and it then points to a condition of considerable gravity. For some reason not yet explained, it is more prone to occur in apical pneumonia than when the lower lobes are involved, though this is denied by some observers.

Old persons are especially liable to a quiet delirium resembling that of typhoid fever.

Persons delirious from pneumonia require special watchfulness on the part of nurses and attendants. Occasionally the patient be-

comes maniacal, and seeks to escape from imaginary enemies. Thus a young man, not alcoholic, in the service of the writer in the New York Presbyterian Hospital, during a momentary absence of the attendant, dashed across the ward and through the sash of a closed window, and fell from the second story to the ground. He recovered from his pneumonia and from the injuries sustained from the fall. In alcoholics this sudden frenzy is of frequent occurrence.

The delirium is not always proportioned to, or dependent upon, the temperature. It may be present when the pyrexia is very moderate. When this is the case it is of evil omen, as it indicates cerebral exhaustion. It may be a question whether the brain symptoms in a given case are the result merely of disturbed cerebral nutrition as in many other forms of disease, or whether they depend upon the direct local action of the microbe. The frequency with which the pneumococcus is found in the fluid of meningitis, even when pneumonia is not present, shows that it has a special predilection for that locality, and it is perhaps more frequently present than we are in the habit of admitting. If autopsies in pneumonia more generally included the cranial cavity, it is probable that more cases of meningitis would be discovered; its distinctive clinical features being masked by those of the primary disease.

When the aëration of the blood is seriously interfered with, we have in this condition alone a sufficient cause for some degree of mental disturbance, which shows itself more particularly in somnolence or coma.

According to Potain,[16] of Paris, the delirium of pneumonia is the result of varied conditions of the cerebrum; ranging from simple excitement, through the meningitis state, to true meningitis. Following the excited stage there is a period of quiet that deepens into coma, at times associated with signs of compression of the basilar cranial nerves. The meningitis may be apoplectic in character.

Aside from the occurrence of delirium there are other evidences of nervous implication. The most striking, perhaps, is the extreme *prostration* that marks certain cases even in the early stage. It is not uncommon that a patient is stricken down in a way that at once suggests the agency of a powerful infection. His strength leaves him, he takes immediately to his bed, his limbs shake, his tongue is tremulous, his head and bones ache, he is nauseated and perhaps vomits. All this is in addition to the chill and to the stitch, and seems at a stroke to deprive the patient of half his vitality. It is in striking contrast to the comparatively slight impression upon the nervous system which is often noted in cases in which the temperature may be higher and the pulmonary condition much more serious.

Sleeplessness is another condition not at all uncommon. Sometimes for nights and days there is no sleep, unless the patient is overwhelmed with hypnotics. This condition should always suggest a careful inquiry into the previous habits of the patient, for while it is by no means confined to those with alcoholic antecedents, it is much more frequent in such persons, and in the absence of delirium the key to the problem might be overlooked. In subjects not alcoholic, the condition seems to be one of cerebral irritation at first, and later, perhaps, of cerebral exhaustion.

In children often, and very rarely in adults, *convulsions* take the place of the initial chill. As the disease progresses, and particularly in cases in which hæmatosis is seriously interfered with, somnolence, deepening into *coma*, is a very frequent condition. As this tends to favor the occurrence or aggravation of hypostatic congestion, frequent efforts should be made to arouse the patient that he may be excited to more vigorous respiration. Life may hinge upon the observance of this precaution at a critical juncture. Sponging the face, neck, and between the shoulders with ice water is an efficient means of recalling the patient to consciousness under these conditions.

Causes of Death.—In fatal cases death may occur in a variety of ways. First, there are cases in which the patient is overwhelmed by the intense virulence of the infection, death occurring within from thirty-six to forty-eight hours after the chill. It would seem as if all the vital functions were overpowered by the toxæmia. There is extreme muscular and nervous prostration, the heart's action becomes rapid and feeble, digestion is suspended, the kidneys act imperfectly, delirium and coma supervene, and death occurs from acute asthenia.

In other cases death is caused by exhaustion of the right heart. The muscle tires out from overwork, being enfeebled already by the action of the poison. Later, dilatation takes place, perhaps to the extent of producing incompetence of the tricuspid valves. Finally, it becomes so overdistended that it cannot recover itself and asystole results.

Still another cause of death is loss of respiratory surface. This is rarely the result of simple pneumonic consolidation, but there is added to this congestion an œdema of other portions of the lung. In these cases the lungs fill up more and more; the breathing becomes more and more rapid and superficial; and death by asphyxia takes place.

Plicque[17] lays much stress upon this mode of death, declaring that nine patients succumb to it for one dying from the infection.

In a considerable proportion of cases death comes from exhaustion of the vital powers, after a protracted and intense struggle which

the system is unable longer to continue. This is common in feeble and old persons, and is marked by a gradual giving out of the forces. It usually does not occur until after the febrile period.

Lastly, death may be caused by one or more of the many complications to be described hereafter.

In addition to the foregoing, sudden death may occur at any stage of pneumonia, with no previous warning.

As remarked by Wells,[17] cases of this kind caused by heart clot, paralysis of the heart, and apoplexy may be readily explained, but there are cases in which no gross anatomical lesion can be found, and which with our present knowledge cannot be accounted for. Wells[18] quotes a number of such cases. The essay referred to contains a great amount of material that cannot be presented here, but which is extremely interesting.

Physical Signs.

Usually, even before there is notable dulness on percussion, and sometimes within three or four hours after the beginning of the attack, we have a fine crepitant râle over the seat of the lesion. This is a sign commonly regarded as peculiar to pneumonia. It is heard only during inspiration, and often is confined to the last third of the inspiratory act. It has been likened to the crackling of salt thrown upon a heated surface, or to the sound produced by rubbing a lock of hair between the thumb and finger. A still more accurate comparison is with the crackling of a static electric machine when the poles are almost in contact. It is supposed to be produced by the bursting of minute air bubbles in the bronchioles or alveoli. It is not to be confounded with the subcrepitant râle, which is coarser, and is heard in expiration as well as inspiration. The crepitant râle ceases when consolidation takes place, the exuded material then occupying the alveoli and excluding the air. The crepitant râle is absent in a small proportion of cases. It sometimes disappears for a time and then returns, to disappear finally as the solidification becomes complete.[19] This sign is accurately limited to the affected portion of the lung, not being heard outside of the pneumonic area, although not always present in all parts embraced within that area. Indeed, in most cases we fail to find it over a large surface at any one time, though repeated examinations will reveal its presence at different points. This is due to its dependence upon transient conditions that vary within narrow limits with the changes going on in the lung, and to the crepitation not being a sound of sufficient intensity to be heard at any distance from the point at which it is produced.

It may be reproduced when the exudate begins to resolve. Re-

curring during resolution it is known as the *crepitus redux* or *râle redux*. It then is coarser than the true crepitant râle, and partakes rather of the character of the subcrepitant.

As consolidation proceeds the crepitant râle is replaced by a new sound, bronchial or tubular breathing. The French call this the *pneumonic souffle*. It is a high-pitched, whiff-like sound, and may be closely imitated by stroking the coat sleeve lightly with the tips of the fingers. Often it is heard only in expiration, during which the current of air is more rapid than during the slower movement of inspiration. It is produced by the passage of air through the finer tubes, the vibrations being conducted with especial clearness by the solid tissue of the surrounding lung. It simply indicates consolidation, and therefore is not distinctive of pneumonia. At first it merely modifies the vesicular murmur, giving rise to *bronchovesicular* respiration, but as the alveoli become more completely filled it excludes vesicular respiration completely in expiration, and imparts a decidedly bronchial character to the inspiratory sound, which sound is at the same time shortened and incomplete. It is a sound of considerable intensity, and may be transmitted to some little distance beyond the consolidated area, and even over the contiguous portion of the sound lung. In children this is especially the case. When fully developed, bronchial respiration is usually a persistent sign, being found at each examination during the whole period of consolidation. If temporarily absent from a locality where it was formerly found, the absence is due to a transient obstruction of a bronchial tube.

If a considerable area of lung is consolidated and therefore withdrawn from action, a compensatory increase of expansion of the unaffected portion is necessitated, and as a result we have exaggeration of the normal respiratory murmur, the condition known as *puerile* respiration. This may be very marked, not only in the sound parts of the affected lung, but also throughout the other lung as well.

The vocal resonance is modified by the greater density of the medium through which it is conveyed. The sound becomes loud, harsh, and metallic, reminding one of the voice as heard through a telephone. To this sign the term *bronchophony* is applied. In eliciting this sign it is important to cause the patient to speak as much as possible *from the chest*. As this is difficult in the case of women, the vocal signs in them are much less marked and conclusive than in men. Much depends upon the words the patient is required to pronounce. The usual *one, two, three* will illustrate this, the first giving much more decided results than the second and third. It is to be borne in mind that the normal vocal resonance is more pronounced on the right side than on the left. In some cases *pectoriloquy* is developed,

that is, the words spoken are heard as distinct articular speech, when the ear is applied to the chest. Bronchophony is not developed until the second stage, as it depends upon solidification of the lung. In the normal condition the sound vibrations formed in the bronchial tubes are greatly softened, or almost suppressed, by the spongy lung tissue interposed between the tubes and the chest wall. When this tissue becomes solid by the filling up of the air cells, the bronchial sounds are transmitted directly to the surface, and seem to be formed immediately under the ear of the auscultator. This apparent near-

FIG. 1.—Stethoscope for Auscultation of the Posterior Portion of the Chest, the Patient Being in a Recumbent Position.

ness of the sound, which is sometimes almost startling, is quite as characteristic of bronchophony as is the modified character of the sound itself.

Like tubular breathing this sign is merely significant of solidified lung, and therefore does not necessarily indicate the presence of pneumonia. In connection with other signs and symptoms, however, it is of great diagnostic value.

Along with bronchophony goes *increased vocal fremitus*. This may be appreciated by placing the palm upon the chest, or more delicately by pressing lightly with the finger tips in the intercostal spaces. In cases in which there is abundant pleuritic exudation over the pneumonic lung, the fremitus is less exaggerated.

Very often the sounds of the heart are heard over a greatly increased area, being conveyed to a distance by the consolidated lung.

In pneumonia it is often difficult to auscultate the posterior portion of the chest. To turn the patient so as to bring the affected side uppermost will frequently cause extreme respiratory distress; and with a weak heart it is not wise to raise him into a sitting posture. To meet this difficulty I have devised a stethoscope (Fig. 1) that, by pressing down the mattress with the left hand, can be slipped under

the patient on the fingers of the right. The cup of the stethoscope is flat and shallow, in shape like the cover of a pill box; and the rubber tubes instead of coming off from the top, come off from the side at points but little removed from each other. The thickness of the cup is about half an inch. With this instrument the auscultatory signs can be obtained with scarcely any disturbance of the patient.

Very early in the progress of the case a degree of *percussion dulness* is observed. At first it is very slight, and is appreciated only by comparison with the note obtained over the corresponding location on the sound side. But it soon becomes more pronounced, and when the consolidation is fully developed the percussion note is but a shade removed from absolute flatness. Beginning in a portion of the lobe, it spreads usually until the entire lobe is included. Often it is pronounced at first only at a central point, from which the dulness fades away in all directions, but as time goes on the area increases until it accurately represents the contour of the lobe. If a second lobe becomes involved it is rarely by continuity, most commonly by the formation of a second distinct focus.

In some instances, however, the vesicular resonance is replaced by a sound which is more or less tympanitic, *skodaic resonance*. It is high-pitched and wooden in character, as when one taps with the fingers on a table. The contrast is marked with flatness on the one hand, and with the sonorous resonance of pneumothorax on the other. This type of resonance is produced in the bronchial tubes within the consolidated area, the air within these tubes being caused to vibrate by the impulse conveyed to it through the solidified lung, and the vibrations being transmitted in turn through the same medium to the surface. Over the larger bronchi the cracked-pot sound may occasionally be elicited, the shock of the percussion stroke being transmitted in the same manner.

Along with the dulness on percussion, or the peculiar form of tympany, as the case may be, there is a well-marked sense of resistance imparted to the finger over the consolidated area. This will serve to correct any erroneous inference that might be drawn from an unexpectedly resonant percussion note. This sense of resistance is often more appreciable than the actual percussion dulness.

With regard to all these physical signs, however, it is to be borne in mind that when the consolidation begins centrally in the lobe it may not be possible at once to obtain the usual results of auscultation and percussion, the intervening normal lung preventing the abnormal sounds from reaching the ear, and at the same time giving out a clear note on percussion. In such cases we have to depend for a while on the rational symptoms for a diagnosis. But it is not as

a rule more than one or two days before the physical signs begin to be apparent, and in the end we may have them as clearly presented as under the usual conditions. This central beginning with the delayed physical signs is very apt to occur in patients advanced in age, or in whom, from any cause, the vital powers are not active.

In the aged the physical signs lose somewhat of their distinctiveness. There is a tendency to increased resonance on percussion due to the rigidity of the bony framework of the chest, the greater depth of the thorax, and the backward curvature of the spine. The calcification of the rings of the trachea and bronchial tubes makes both the percussion note and the voice sound more sonorous, while it impairs the distinctive character of vesicular breathing, substituting a somewhat bronchial quality. The excess of the bronchial secretion, which is almost physiological in old age, is likely to obscure the crepitant râle, which, in any case, is frequently absent in senile pneumonia.

Auscultation of the Right Heart.—In a considerable proportion, perhaps more than half, of the cases of pneumonia, we have sooner or later to confront what may become a very serious mechanical condition—the overfilling of the right heart and the venous system. This condition has its appropriate signs and symptoms, in speaking of which I shall use to a considerable extent the language of a former communication.[20]

Whenever there is obstruction of the pulmonary circulation, the labor of the right heart is necessarily increased. In proportion to its inability to overcome the obstruction, there will be an accumulation of blood in the venous system. Excess of blood in the veins implies deficiency in the arteries, and hence this class of cases is characterized by an unequal divison of the blood between the venous circulation and the arterial.

This condition has extremely important consequences, especially in acute pulmonary affections. In these we study the pulse with the greatest solicitude to judge how the heart, as we say, is supporting the struggle. But the arterial pulse gives no indication of the immediate peril, for it is not the left heart that is bearing the brunt of the battle. The pulse tells its story only at second-hand. It may be small and weak, but it is chiefly because the left heart does not receive enough blood from the lungs to fill its chambers and to distend the arteries. The trouble is not in a lack of propelling power so much as in deficiency of blood to be propelled.

But if, instead of feeling the radial pulse, we could lay our finger upon the pulmonary artery, we should obtain information vastly more to the point. We should then be able to appreciate the degree of pulmonary obstruction by the fulness of the vessel, and to rate the

power of the right ventricle by the force of the arterial beat. And in the relation of these two factors one to the other is involved the issue of the case. Increasing obstruction with decreasing right-heart power means death; decreasing obstruction with sustained right-heart power gives promise of recovery. It is a question with which the left heart, and therefore the radial pulse, has almost nothing to do. For the peril is not from general exhaustion, as for example in fever, nor from failure of the heart as a whole, as in some cases of infection, but specifically from tiring out of the *right* heart in its effort to unload the venous circulation through the obstructed vessels of the lungs.

Now, while we cannot place our finger upon the pulmonary artery, we can obtain nearly the same information by applying the stethoscope over the pulmonary valve. Owing to anatomical conditions which it is not necessary to describe here, it is entirely practicable to separate the pulmonary valve sounds from the aortic, and by means of auscultation to study the peculiarities of the former as indicating the condition of the pulmonary circulation.

Unfortunately, however, in some cases the valve sound is masked by bronchial râles, so that it may be impossible to appreciate it accurately. But even in the most rapid breathing there are brief intervals during which the practised ear may nearly always gather the required information.

Now, if we note carefully the sound of the pulmonary valve in, for example, a case of pneumonia, we shall find that at the outset, while the right ventricle is still in vigorous action, this sound is especially clear and sharp, indicating a quick and strong recoil of the pulmonary artery following the ventricular systole. This sharp recoil is due to unusual distention of the vessel, and this in turn is due to the resistance which the blood meets in passing through the lungs.

If the case is to terminate favorably, this accentuation of the pulmonary sound will probably continue through the whole course of the disease, becoming less marked as the obstruction in the lung decreases. But in cases of increasing severity and with an unfavorable tendency, a time soon comes when not only this accentuation is lost, but the normal intensity of the valve sound is lessened, the sound becoming weaker and weaker until it ceases to be heard. This means, not that the obstruction has become less, but simply that the muscular power of the right ventricle has become exhausted with the labor exacted of it. The blood is no longer driven through the artery with sufficient force to distend it, and there is not enough recoil to bring the valve cusps together with an audible sound.

When this point is reached, the end is not far off. The weaken-

ing of the right heart favors still greater pulmonary obstruction, and this in turn adds to the burden of the right ventricle, thus completing the vicious circle. The struggles of the ventricle become feebler and feebler, while the tension within its cavity constantly increases, as the blood presses into it from behind. At last there comes a moment when the overtaxed muscle cannot summon the energy for another contraction, and its action ceases in diastole.

The steps which lead up to this result are in a great degree traceable by symptoms and physical signs. First of all, there are auscultatory and other signs of pulmonary obstruction; then come signs of general venous congestion. The distended right auricle may be traceable by percussion, or even may be seen pulsating at the right of the sternum. An increased area of cardiac dulness extending towards the xiphoid cartilage indicates the repletion of the right ventricle, and in spare subjects the labored beating of this may be felt by pressing the fingers under the costal cartilages. The superficial veins are seen to be unusually prominent, and the liver is enlarged. The spleen also is increased in size, and evidence of intestinal congestion may be afforded by copious diarrhœa.

Proof of passive hyperæmia of the kidneys is found in lessened excretion and in albuminuria. Thus all things combine to indicate a general preponderance of blood in the venous side of the circulation, the result which we should naturally expect from a retardation of the blood in the pulmonary vessels.

Clinical Types.

Bearing in mind that in a very large proportion of the community the potential cause of pneumonia is already present in the pneumococci so often to be found in the air passages, it is easy to understand that a great variety of circumstances may act as exciting causes and determine an attack. Under normal conditions the vital forces are sufficient to protect the individual against the microbic action, but any influence which impairs this defence may open the way to effective invasion. These influences present a great variety, and it has been the custom with some writers to classify pneumonia according to the condition which seems to have given rise to it. Thus, we have bilious pneumonia, grippe pneumonia, malarial pneumonia, typhoid pneumonia, rheumatic pneumonia, traumatic pneumonia, etc., etc. With our present knowledge it is clear that these are not varieties of the disease, but merely designations of the agency by which in any particular case the defences of the system have been broken down and the specific germ afforded an opportunity to act. In some

instances there is a mingling of the phenomena of the exciting cause with those of the induced pneumonia, as when the fever under the influence of malarial infection takes on a remittent type, or the presence of tubercle in the lung complicates the physical signs or changes the typical temperature curve.

But while it does not seem that any useful purpose would be served by adopting such a classification and describing the various forms into which pneumonia may thus be divided, yet the prominent features of the disease vary in different cases, and the variations for the most part group themselves into three divisions, more or less marked, constituting three general types which shade into each other. In a clinical lecture by the writer, published in 1895, and in which these types are described the following passages occur.[21]

"To-day it is generally recognized that pneumonia is a disease of microbic origin, and that while the direct action of the microbe is limited in a greater or less degree to the lungs, the toxin derived from it pervades the entire system.

"The local lesion is not necessarily in proportion to the systemic infection; that is, we may have a very large implication of the lung with comparatively little evidence of general infection, and conversely we may have severe general infection with but little pulmonary implication. And again, without reference to the amount of lung involved, or even to the degree of fever present, we may have the vital forces but little impaired, or we may have the nervous and muscular systems completely overpowered by the intensity of the poison, constituting a condition that might almost be termed malignant.

"Giving to terms already in use a more definite meaning than they have had heretofore, we may call these two classes of cases *sthenic* and *asthenic*. We may also admit a third division, based upon mechanical conditions by which the pulmonary circulation is rendered especially difficult, and to this division the term *obstructive* may be applied. Viewed then from our present standpoint we may recognize three types of cases of pneumonia: sthenic, asthenic, and obstructive.

"It so happens that we have in the wards at this time patients illustrating these three divisions.

"The first case I will show you is that of a man twenty-eight years of age, of previously good health, and temperate in his habits; he is now in the fourth day of the disease. His initial chill, four days ago, was of moderate severity, the pain in the chest was considerable, the temperature rose on the second day to 103.5° F., and his pulse to 105, and on the following day the temperature reached 104.5° F., and his pulse 116. At this time the respirations were 32 per minute.

There is in this case a remarkable absence of the prostration which we see in many cases of pneumonia. The patient, on arriving at the hospital, insisted upon walking from the carriage to the elevator, and from the elevator to his bed in the ward. You will perceive now that when he is asked to sit up for physical examination of the posterior portion of the chest, he rises without any evidence of feebleness; his hands are perfectly steady, and when his tongue is protruded there is no tremor. He is entirely capable, if allowed to do so, of walking about the ward. His mind is perfectly clear. When asked how he feels, the reply is 'first-rate.' On examination, we find that on the right side, with the exception of the space above the fifth rib in front, and above the middle of the scapula behind, there is evidence of consolidation over the whole lung, that is to say, we have dulness on percussion and tubular respiration. Now, in this case, we have a large mass of lung implicated, while at the same time the constitutional symptoms are relatively slight. We are justified, therefore, in considering that in this case the force of the infection is comparatively moderate, and that the local lesion is the more prominent factor. On examining the heart we find that the pulmonary second sound is decidedly accentuated, indicating that there is marked obstruction to the pulmonary circulation on the one hand, and, on the other, a vigorous action of the right side of the heart. The blood is propelled into the pulmonary artery with force, but meeting resistance in the pulmonary circulation, there is corresponding recoil of the column of blood against the valve, causing a loud and sharp pulmonary sound. This patient's urine has been examined, and has been found free from albumin. The test with nitrate of silver shows the usual absence of chlorides.

"The next case that I have to show you is one in which the conditions are materially different from those which we have just considered. This patient is a man, thirty-five years of age, of fairly good previous health, a car driver by occupation. His initial chill occurred three days ago; it was very severe, lasting, as he stated, more than an hour; it was followed by very marked prostration. From the moment of its occurrence there was a feeling of intense weakness; he took to his bed immediately, and, when removed to the hospital, had to be carried from his bed to the carriage, and from the carriage he was taken on a stretcher to the ward, and had to be lifted into bed. You find him now exhibiting the evidence of extreme muscular and nervous prostration. We place a glass of water in his hands, and in attempting to carry it to his lips he seizes the glass with both hands, nevertheless there is so much tremor that a portion of the water is spilled. The tongue is also tremulous. He is at

times delirious, with busy active delirum, a constant desire to get out of bed; but should he accomplish this, he would fall helplessly to the floor. His temperature on admission was 103.5° F., his pulse 132, and his respiration 30. The relative slowness of respiration, as compared with the pulse, shows that it is not the amount of lung implicated which constitutes the gravity of the affection; the pulse being so much more frequent than the respiration indicates that the cardiac ganglia and probably the heart muscle are directly affected by poison. On examining the heart we find that the first sound is extremely feeble, and the pulmonary sound is almost inaudible. The pulse is very small as well as frequent. It is stated that at the onset, immediately after the chill, there was vomiting. These conditions together indicate a very grave implication of the nervous system, showing an intensity of infection in marked contrast with that of the previous case. The urine also is found to be albuminous. On physical examination, we find on the right side dulness and tubular breathing in the summit of the lung, extending as far down as the fifth rib in front, and nearly to the angle of the scapula behind; in other parts of the lung there are a few moist râles, and also occasionally throughout the left lung. Still, the evidence of the physical signs is that the action of the respiratory apparatus is but moderately impaired. The gravity of the case depends upon the virulence of the infection, the direct poisoning of the nervous and muscular systems. The muscular weakness, which is so marked elsewhere, extends to the heart, as is indicated by the small and very frequent pulse, and by the character of the first sound. It is in this that the danger to our patient lies; it is not that his respiratory function is so far impaired as to create peril in that direction, but the danger is primary heart failure, due to the poisoning of the nervous system and of the muscular fibre. The whole heart in this case, both the left as well as the right side, is markedly feeble, and in our treatment the effort must be to keep up the cardiac action until the force of the infection is spent. Cases of this type are more apt to prove fatal during the stage of pyrexia. If we can tide them over until defervescence takes place the prognosis becomes much more favorable.

"Our next patient is a man sixty years of age, whose habits have been irregular, and whose constitution has been broken down by alcohol. He is now in the sixth day of the disease; the chill was not well pronounced; there was but moderate pain in the chest, but early in the case the difficulty of respiration became a prominent factor. We find him now with a temperature of 102° F., a pulse of 130, and with 48 respirations to the minute. The face is pale, the lips are blue, and the superficial veins are distended. On physical examina-

tion, we find that the lower lobe on the left side is for the most part consolidated; we find also that there are abundant mucous râles and perhaps a slight dulness throughout the remainder of the left lung, and also through the right lung. We have, therefore, a condition of pneumonic consolidation in the lower lobe of the left lung, with œdema more or less pronounced in the remainder of that lung and throughout the other. This condition necessarily implies a very grave impairment of the respiratory function. The amount of air which finds its way to the pulmonary vesicles is reduced to an extent which seriously threatens death by asphyxia. On examining the heart, we find by percussion that the right chambers are distended, the area of cardiac dulness being increased in the direction of the sternum. The pulmonary second sound is extremely feeble, being scarcely audible, obscured as it is by the mucous râles in that locality. The examination shows an increased area of hepatic and of splenic dulness, indicating that both the liver and spleen are distended with blood. The urine is albuminous. In short, we have everywhere the evidence of venous repletion. There is more blood in the veins and less in the arteries than in the normal condition. The obstruction to the pulmonary circulation calling upon the right ventricle for increased action, that portion of the heart is especially exposed to exhaustion.

"The leading factor in this case, therefore, is the impairment of the respiratory function with consecutive exhaustion of the right side of the heart. The danger is that the right heart will fail. And yet the original area of pneumonic consolidation was not great, not nearly so great as in the first case we examined. The difference lies in the condition of the circulatory apparatus, due, in the first instance, to the age of the patient and, in the second, to the alcoholic habit which has impaired the whole mechanism of circulation. With a vigorous heart and sound blood-vessels, the amount of pulmonary consolidation present would have constituted but a trifling danger. We have not in this case, either, the evidences of virulent infection, and the nervous system is not markedly implicated.

"When death occurs in such conditions the post-mortem shows the right cavities and the pulmonary artery distended with blood. In a private note which I received a few days ago from Dr. Daland, of Philadelphia, he describes the post-mortem appearances in four cases of this kind which he had observed. The right auricle and ventricle and the pulmonary artery were crowded full with a substance resembling currant jelly. The tendency to an early unfavorable termination in these cases is not so great as in the preceding type. Defervescence is not so marked nor so complete, and its occurrence does not affect

the prognosis so favorably. The mechanical conditions are but little affected by the temperature, and death often occurs when the pyrexia is very slight."

In addition to the foregoing types there may be differences in the clinical features of the disease depending upon mixed infections. Other organisms may be present with the pneumococcus lanceolatus, such as the pneumococcus of Friedländer, Pfeiffer's influenza bacillus, the Klebs-Loeffler bacillus, the typhoid bacillus, the staphylococcus pyogenes, and the streptococcus. One or more of these acting with the diplococcus proper to pneumonia may modify more or less the clinical picture.

The one most likely to take part is the streptococcus. It may be present from the beginning of the attack, or it may become associated at a later stage. If present from the first, the onset is likely to be less violent than usual, and the invasion more tardy in its movement. The sputum is more purulent, less vitreous, less sticky, and contains both organisms. The face is less flushed and has the aspect of sepsis. The temperature is irregular, and there is no crisis. Lysis may be extended over three or four weeks.

The upper lobe is more liable to be attacked, but the points of invasion change frequently, and the physical signs persist, perhaps for five or six weeks or longer. The cough is troublesome and there is great prostration. It is often months before the health is regained. And yet, with all this, the percentage of mortality is rather less than in pure pneumococcic infection.

Weismayr reports 39 cases of pneumonia in which the pneumococcus lanceolatus was found alone in 34, and associated with the streptococcus in 2, while in 3 the streptococcus was found alone. Of the 34 cases of pure pneumococcus infection 27 defervesced by crisis, 4 by lysis, and 3 proved fatal on the seventh, tenth, and twelfth days respectively, defervescence never being later than the eleventh day, excepting in 1 case when it was completed on the fifteenth day.

In contrast with this is the behavior of the five streptococcus cases. Of these one was fatal on the nineteenth day, and in the remaining four there was kept up an irregular remittent fever, lasting in one case until the thirty-seventh day. The signs of consolidation continued from nineteen to forty days.

The diagnosis of streptococcic pneumonia is based upon the atypical progress and long duration of the disease, but can be reached with certainty only by microscopical examination of the sputum.[23]

Pathology.

Autopsical Findings.

These correspond to a process extending from simple hyperæmia of the affected part through extreme engorgement, fibrinous and cellular exudation into the air cells, complete consolidation, fatty degeneration of the exudate, and removal of the latter by absorption and expectoration. All of these stages may be represented at the same time in different portions of the lung. As to how far these changes correspond with the clinical phenomena of the disease, and the results of auscultation and percussion, it will be interesting to quote from a work which is at present a leading authority on the subject of pneumonia:[2]

"We may pause here to consider whether in a disease clinically so well defined as pneumonia, it be not possible to assign correctly the special anatomical changes which accompany the various periods or stages of the disease, such as are denoted by the pyrexia and physical signs. It is undoubtedly true that this can be done to a certain extent, but a little consideration will show a lack of absolute parallelism between the anatomical and clinical features. At the outset, the latter denote the existence of a more or less widespread area of congestion in the lung, as well as of incipient pleural inflammation, which are conditions of structural change possible to be detected in the dead body. Yet there must be a condition antecedent to these changes that constitutes the so-called 'first stage,' for which there is no anatomical indication. Indeed, as we have seen, the stage of pulmonary engorgement has seldom been clearly demonstrated anatomically; for the obvious reason that at this early period of the affection death rarely occurs, and also because in cases which run the most rapidly fatal course their very intensity carries them beyond this stage before death. And as has been pointed out, it is extremely difficult to infer from post-mortem appearances the existence of ante-mortem congestion in any organ, and especially in an organ like the lung, which is so readily influenced by the circulatory changes that arise in the last hours of life, or in the act of dying. It is well known how at this time the blood tends to accumulate in the venous system, and to be especially prone to collect in the dependent parts from the failure of the heart to propel it onwards; and this resulting condition of hypostasis is even more liable to occur when the heart is already weakened by the febrile process. The lung may become so filled with serosanguinolent exudation as to be quite solidified, and the term 'hypostatic pneumonia' has been employed to denote this."

The earliest lesion is simple hyperæmia. When the chest is opened in a case in which death has occurred from pneumonia a portion of the lung may be found in this condition. It is seen not to be so fully collapsed as the surrounding normal lung, and to the touch it is slightly more resistant. The color is bright red, modified by the natural pigmentation of the lung. The pleural surface has lost some of its brilliancy, the epithelium being cloudy or perhaps exfoliated. On section, the surfaces are bright red, and exude a bloody frothy serum. The physical sign corresponding to this condition is scarcely more than a slight localized feebleness of respiration, with more or less abundant moist râles. A few hours later, the hyperæmia has passed into an extreme degree of vascular engorgement. The diseased part shrinks but little when the chest is opened. The pleural surface is of a deep red color, veiled by more or less of fibrinous exudate, which peels off readily in flakes. A similar condition is often observed on the corresponding costal surface. The resistance to the touch is markedly increased, and on section the tissue is more readily divided than in healthy lung. The cut surfaces are dark red in color, and dark blood mingled with air follows the knife. The appearance resembles closely that of an incised spleen, and the term splenization is applied to this stage of the local disease. At points minute extravasations of blood may be observed in the substance of the engorged tissue. Microscopically, the pulmonary capillaries will be found distended with blood, more or less fluid occupying the air cells.

But the lung still crepitates between the thumb and finger, and can be squeezed dry without breaking down. A piece of it thrown into water floats.

The physical signs belonging to this condition of the lung are diminished resonance on percussion, bronchovesicular respiration, crepitant râles, and slightly increased vocal resonance. In addition a pleural friction sound is often present.

The distinguishing feature of the next stage is the filling up of the air cells with a fibrinous exudate by which the parenchyma is completely solidified. If a considerable area is involved the lung is increased in bulk and pushes out into the intercostal spaces, so that furrows are imprinted on its surface by the ribs. The lung is usually covered by a dense white, or grayish, or yellowish layer of fibrin, which if removed shows a deep mottled red or purple color beneath. This layer is adherent to the costal pleura also, and the separation leaves both the pulmonary and the costal surfaces rough and irregular. Osler[23] states that the pleural exudate may form a creamy layer an inch in thickness. When cut into, the solidified mass is found to

have much the consistence of liver, and to be dark red or brownish-red in color; and for this reason the condition is known as *red hepatization*. In asthenic cases the proportion of fibrin in the exudate seems to be less, and the hepatized portion is flabby as compared with what is seen in cases belonging to the sthenic type. The cut surface has a granular appearance due to the projection of the little

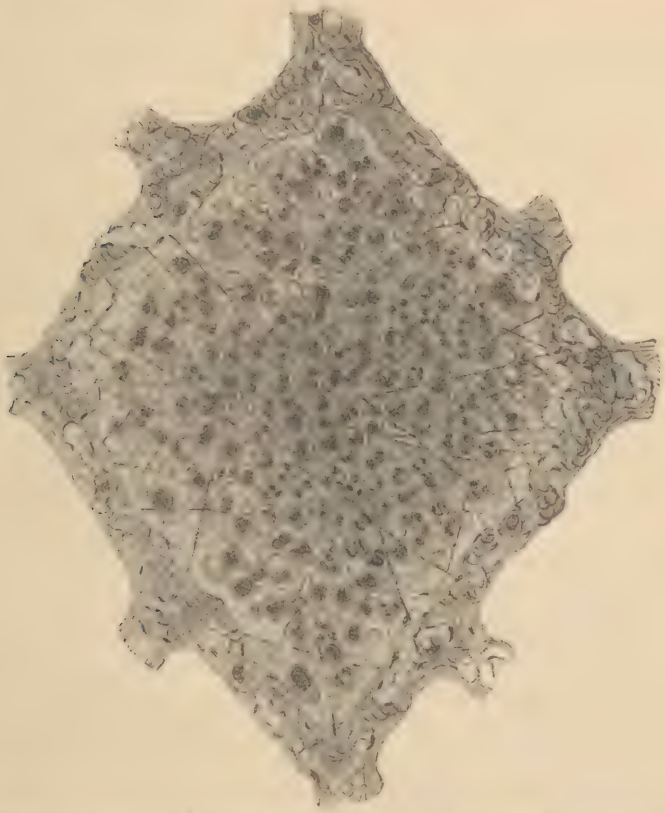

Fig. 2.—Air-cell Filled with Pneumonic Exudate. (Drawn by Dr. Louise Cordes from a specimen in the laboratory of the Presbyterian Hospital, New York.)

plugs of fibrin with which the alveoli and the bronchioles are filled. These can readily be scraped from the surface of the section, and are found to be casts of the air cells intermixed with cylindrical moulds of the ultimate divisions of the air tubes. The infiltrated tissue does not crepitate on pressure, is friable, and can be torn much more readily than normal lung structure. Where a bronchial tube is cut across a sticky mucus of variable color flows out. A piece of hepatized lung sinks in water.

During this stage the weight of the lung may reach 2,500 or 3,000 gm., instead of 600 gm. which is the normal average.

Microscopically the moulds of the alveoli already alluded to are found to consist chiefly of fibrin in the form of a delicate mesh, to which are added red blood corpuscles, leucocytes, epithelial cells, and numerous diplococci pneumoniæ. Other organisms, such as streptococci, staphylococci, etc., are often found associated with the specific microbe.

As this stage approaches completion the capillaries derived from the pulmonary artery become occluded, and the resulting thrombosis extends backwards into the larger vessels, even as far as to the giving off of the branch supplying the lobe. Indeed, there are cases on record in which the thrombus has reached the bifurcation of the pulmonary artery itself, and a portion of the clot has broken off and been swept into the sound lung, causing immediate death.

This stoppage of the functional capillary circulation is perhaps due in part to vital changes growing out of the microbic invasion, but the chief agency is the pressure exerted by the exudate. The tension in the pulmonary circulation being scarcely half that in the systemic, a comparatively slight mechanical cause is sufficient to arrest the movement of the blood.

The septa between the air cells are but little changed beyond the above-mentioned engorgement of the vessels and perhaps some loss of epithelium. In their migration from the capillaries some of the leucocytes remain entangled in the stroma. The nutritive vessels remain pervious.

The pleura at this stage is usually covered by a dense layer of false membrane, which dips also into the interlobar fissures, and bridges them over. This layer of fibrin very generally extends beyond the area of hepatization, becoming thinner towards the edges of the patch. It may cause an adhesion of the two pleural surfaces, which latter, however, are easily torn apart.

The physical signs indicating the existence of hepatization are dulness, approaching to flatness, and increased resistance on percussion, with tubular breathing, or perhaps entire absence of respiratory sound if the exudate extends far into the tubes. To this is added increase of vocal resonance and of vocal fremitus.

Succeeding the stage of red is that of *gray hepatization*. The change results from the absorption and removal of much of the coloring-matter of the red cells in the exudate, the addition of white corpuscles in great number, and a disintegration of the formed elements comprised in the contents of the air cells into a fatty, granular material. As the term indicates, the color of the cut surface is changed

from red to gray, but as the change takes place irregularly the color is more or less mottled. In consistence the tissue is softer than in the previous stages, so that the finger may be thrust into it, and the pit thus formed fills with a dirty puriform fluid. This becomes more marked as the change progresses, and the cell contents become more liquid. The term purulent infiltration is applied to this condition incorrectly, as the fluid is not pus but a fatty and granular detritus.

Some confusion, however, has arisen in regard to the condition to which the term *purulent infiltration* properly belongs. By some this is considered as merely an advanced stage of gray hepatization, the fibrin and the red cells in the exudate having broken down and the leucocytes undergoing fatty degeneration. These authorities regard the condition as a step in the progress toward resolution, a preparation of the exudate for being absorbed.

Other authorities describe purulent infiltration as one of the issues of pneumonia, and almost necessarily fatal. To them it means a diffuse suppuration of the affected portion of the lung, an event so grave as practically to preclude recovery.

It is not difficult to see how this confusion has arisen. Necessarily our knowledge of the local conditions has been obtained from the post-mortem findings, and these may admit of either interpretation. But it seems to me that there are two distinct processes, the results of which may be differentiated both by the clinical history and by the changes found in the lung.

We may find a subject whose expectoration had gone through the usual changes and become mucopurulent, and who had passed the crisis, but has not been able to rally afterwards. Death has taken place from mere exhaustion. The autopsy shows the cell walls intact, and the cells filled with the grayish material, which gives the name to this stage of hepatization.

In another case the creamy sputa will have made their appearance in due sequence, and soon afterwards there will have been a more or less complete defervescence, but the temperature has not remained down. There have been fluctuations resembling those of hectic, with chilliness and perspirations. After a course of several days death has taken place under these septic manifestations. The autopsy shows the affected area filled with a creamy fluid identical with pus, and the septa infiltrated, softened, and more or less broken down.

Now in the first case death has overtaken the patient in the midst of a process tending to restoration to a normal condition. In the second, the restorative action had been interrupted by a new process with its seat in the cell wall. The nutritive vessels have been at-

tacked, and there has supervened a veritable "inflammation of the lungs," going on to suppuration and breaking down of tissue. To this condition the term purulent infiltration is very applicable, and it will be readily apparent that such extensive suppurative action involving corresponding loss of lung substance and coming upon the heels of an exhausting pyrexia could scarcely be recovered from.

There is no physical sign by which the transition from red to gray hepatization is indicated. If resolution begins in this stage it may be marked by a return of the crepitant râle. But resolution may take place before this stage is reached, and in that case the return of crepitation would be during red hepatization.

As to the time at which these several stages develop the greatest diversity obtains, and in this connection the interest of the following note is such that no apology is necessary for its insertion:

"As illustrating the variability of the anatomical characters of the inflamed lung at the time of death, and the lack of any definite relation of these to the time at which death has occurred, the following cases may be quoted, in which death took place at periods varying from six to seventeen days from the onset.

"1. Male, aged 40, death on sixth day. Gray hepatization of greater part of left lower lobe.

"2. Male, aged 75, death on seventh day. Gray hepatization of anterior part of upper lobe (congested posteriorly) and of upper one-fourth of lower lobe of right lung. In left lower lobe a 'recent' pneumonic nodule.

"3. Male, middle-aged, death on seventh day. Whole of lower and part of upper lobe of left lung hepatized; gray at base, elsewhere red.

"4. Male, aged 4, death on seventh day. Gray hepatization and 'purulent infiltration' of right upper lobe.

"5. Male, aged 62, death on eighth day. 'Red passing into gray' hepatization of right lower lobe.

"6. Male, aged 50, death on eighth day. Gray hepatization of right lung (except apex).

"7. Male, aged 24, death on ninth day. Gray hepatization of left upper and lower lobes. Patches of 'reddish-gray' hepatization in right upper and lower lobes. (Fibrinous coagula in bronchi.)

"8. Female, middle-aged, death on ninth day. 'Grayish-pink' (early gray) consolidation of right upper and lower lobes.

"9. Male, aged 48, death on tenth day. Right lung, except extreme apex and anterior margin, entirely in state of gray hepatization.

"10. Female, aged 4, death on the tenth day. Lower and part of upper lobe of right lung in state of 'reddish-gray' hepatization.

"11. Male, aged 18, death on thirteenth day. Both lower lobes 'dark colored and granular.' (This case was rheumatic, and was accompanied by peri- and endocarditis.)

"12. Male, aged 50, death on sixteenth day. Gray hepatization, studded with yellowish points, of whole of right lung, except anterior and lower margins. [Probably consecutive "pneumonitis" A. H. S.]

"13. Male, aged 4, death on seventeenth day. Gray hepatization of left lower lobe." (Sturgis and Coupland.[3])

According to Delafield and Prudden,[21] the lung is found passing from red to gray hepatization at any time between the second and the eighteenth days of the disease. It is found completely gray at any time from the fourth to the twenty-fifth day. In about one-half of the fatal cases death takes place in the condition of red and gray hepatization; in about one-fourth in the condition of gray hepatization.

As resolution progresses the infiltrated material is gradually removed, and little by little the air regains access to the alveoli. The lung tissue becomes less friable, shrinks in volume, is more elastic, crepitates again on pressure, and resumes a more natural color. Corresponding with these changes the physical signs which marked the invasion reappear in reversed order. Percussion dulness becomes less absolute, and bronchial breathing, if previously all respiratory sounds were absent, returns for a time, and then gradually gives place to vesicular respiration accompanied at first by crepitation. Vocal resonance and vocal fremitus are less marked. It is a long time, however, before the physical signs get to be entirely normal, especially if the fibrinous layer covering and uniting the pleural surfaces is of considerable thickness, thus dampening the percussion note and obscuring the auscultatory sounds.

In most cases besides the specific pneumonic lesion, which is limited to a certain area, there will be found evidence of congestion in other parts of the same lung, and perhaps in its fellow. This congestion may be so intense, and the resulting secretion so abundant, as to leave insufficient breathing surface, and to be the immediate cause of death. The congestion may be hypostatic, when it will be limited to the dependent portion of the lung, or it may be due to cardiac weakness, and affect all parts. Very frequently more or less pulmonary œdema is present.

In rare instances the affected portion of the lung undergoes a suppurative process. The surface has then a yellowish color, and on section a purulent fluid exudes. The cells, resembling pus cells in all respects, not only fill the alveoli, but infiltrate the interalveolar walls.[22] The pressure thus induced interferes with the nutrition of the septa, and may lead to their softening and breaking down. This may result in the formation of abscesses, but the condition if at all extreme usually proves fatal before this point is attained.

Abscess of the lung as an event of pneumonia occurs in between one and two per cent. of all cases. The abscess may have firm walls, or may be only an irregular cavity in broken-down tissue. Abscesses vary in size from that of a pea to the dimensions of the entire lobe.

They sometimes discharge through a bronchus, at other times, when small, they become encapsulated and undergo caseation.

After death from pneumonia a characteristic condition of the heart is usually found, in which the left cavities are nearly or quite empty while the right cavities are distended by firm coagula that often extend into the branches of the pulmonary artery.

The spleen is often enlarged, especially in asthenic cases in which the infection has been intense. Under like conditions the liver is congested, particularly when the respiration has been greatly embarrassed and the right heart overtaxed.

In the kidneys the cells lining the tubes are often in the condition of cloudy swelling, and in a small proportion of cases there is fully developed nephritis.

There is a form of pneumonia[2] in which the air cells, instead of becoming entirely free as the result of resolution, are found to contain more or less of connective tissue springing from the cell wall and in intimate connection with it. This is not uniformly disseminated through the air vesicles, some of these containing only fibrin and various cells. The connective tissue may become organized, the new vessels communicating with the vessels of the septa.

The clinical history is somewhat different from that of ordinary pneumonia, being more protracted, and the phenomena of crisis are not well marked. The process realizes the idea of a true pneumonitis. If life continues for several months, the air spaces become completely filled and their walls much thickened; and smooth connective tissue takes the place of the natural structure of the lung.

Pathology.

It is now very generally conceded that the essential phenomena of pneumonia are due to the action of one or more forms of bacilli. In nearly every case the diplococcus pneumoniæ of Fränkel is found in the exudate, and it may also in rare cases be disseminated through the system more or less generally. With this are sometimes associated other microorganisms in such number as to suggest the probability that they play an important though subsidiary part both in the local process and the general infection.

As to the relation of the specific organism to the disease as a whole, we may note:

First, that no amount of traumatism inflicted upon the lung, be the methods ever so varied, produces pneumonia. We may cut, pierce, bruise, burn, or scald the lung; we may introduce mechanical or chemical irritants into the air passages, and while we get inflammation as the result we do not get pneumonia.

Second, we may have pneumococcic infection in several serous and synovial cavities at the same time, resulting in suppuration in each, and not have pneumonia.

Third, we may introduce the pneumococcus into any portion of the body save the lungs, and even into the blood itself, and we do not get pneumonia.

Fourth, but if we introduce active pneumococci into the parenchyma of the lung we always get pneumonia as the result.

Fifth, in probably every case of pneumonia coming to autopsy during the active stage, if the search is properly conducted, the presence of pneumococci in the lungs can be demonstrated.

The inference from these facts is that the one thing necessary for the development of pneumonia is the presence in the alveoli of pneumococci in a condition of active multiplication.

In regard to the nature of the local lesion, while it is commonly designated as an inflammation, the term is scarcely correct in the sense in which it is understood when applied to that process as it occurs in other structures. We consider inflammation as affecting the tissue itself, and causing an anatomical change in its elements. But in pneumonia the interalveolar septa seem to be but little affected by the enormously active process taking place within the cells. After a few days these cells are emptied of the mass of exudate with which they were filled, and the cell walls are found practically in their normal condition, having suffered at the most only a little loss of epithelium. Had this been an ordinary inflammation, as of the liver or the kidneys, accompanied by a corresponding amount of local change, we should expect to find such damage to the part as would require a long process of repair, if, indeed, complete recovery could ever take place. Vessels would be obliterated, new connective tissue would be formed, permanent indurations would remain, etc. Indeed, such changes are the usual accompaniments of chronic pulmonary inflammations, and sometimes remain after pneumonia of an aberrant type. That they do not occur in typical croupous pneumonia seems sufficient evidence that the process in the latter case is essentially different from inflammation in general.

But if other evidence were needed it is supplied in the fact that at the crisis the fever, which if the process were an inflammation we should have to regard as an inflammatory fever, ceases abruptly, while the so-called inflammation goes on. In typical cases there comes a time, usually from the fifth to the eighth day, when the temperature falls suddenly nearly or quite to the normal line, while the physical signs denote that the local conditions remain unchanged. In what inflammation occurring elsewhere, and of sufficient intensity

to occasion a temperature of 103° to 106°, do we find such a defervescence with all the local signs of the inflammation persisting afterwards?

But if this is not an inflammation of the lung tissue, what is it? This question has received various answers. It has been suggested that the fever is the primary affection and that the changes in the lung bear some such relation to it as the rash of scarlatina, for example, bears to the fever that accompanies it. The term lung fever is a survival of this view.

It has even been suggested by Trousseau that the process is essentially a form of erysipelas, modified by the peculiar structure in which it has its seat. And again it has been thought to be a herpes zoster of the lung, having a nervous origin. But even these fantastic suggestions, designed to meet the difficulties in the way of regarding pneumonia as a simple inflammation, still leave the phenomena of the crisis above referred to unexplained.

The discovery of the pneumococcus has carried us along so far as to enable us to say that pneumonia is an infectious disease depending upon the action of this organism. This is a great advance, but it still leaves the question open, How does the micrococcus bring about the local and general conditions? How does it find its way to the lung, and when it gets there, how does it act?

It seems to the writer that there is an answer to this that is at once consistent with the facts and affords a satisfactory explanation of the phenomena observed. It is that instead of an inflammation of the lung tissue we have essentially a process of germ culture going on in the air cells. To make this intelligible, we must recall the fact that in the lung we have two separate and distinct circulations, the one derived from the bronchial vessels, and designed for the nutrition of the structure; and the other derived from the pulmonary vessels, and devoted to the function of the organ. It being chiefly with the latter that the pathological process has to do, while the former is left to maintain the integrity of the tissues, we can see that a diseased and a relatively healthy action may go on side by side, dependent upon the condition of the respective systems of vessels.

Let now a diplococcus find its way into an air cell, and there excite an irritation in the delicate structure that separates the interior of the cell from the functional blood supply. An exudation from the latter follows, and the coccus at once finds itself surrounded by a medium suitable for its multiplication. The conditions as to temperature, etc., are similar to those in artificial cultures in the laboratory. The microbes increase rapidly, more exudate is poured into the cell, which becomes filled and overflows into an adjoining

cell, where the process is repeated, cell after cell and lobule after lobule being invaded. All this time the cell wall is nourished by its nutrient blood supply, and is almost as indifferent to what is going on in the space which it encloses as is the glass of the culture tube to the process within it.

If asked what, after all, is the evidence that the process does not affect both circulations alike, the answer is, that if it did so, the resulting condition would be incompatible with the maintenance of vitality in the affected part. We have, for example, a portion of hepatized lung, perhaps as large as a child's head, in which the branches of the pulmonary artery are thrombosed, and into which no blood from the right side of the heart can penetrate. Suppose for an instant that a like condition existed in the nutrient circulation, that its vessels also were occluded, and that no blood could reach the parenchyma of the lung from the left side of the heart. So large a mass could not be nourished by imbibition from surrounding parts, and there would be nothing left for it but speedy loss of vitality. What actually does happen, however, is, as we have seen, that the cell wall escapes without injury, and not only so, but the absorptive apparatus remains in full activity as is proved by the quickness and completeness with which the removal of such a bulk of material is effected.

From the moment that the culture process begins, the specific toxin also begins to be formed, and is at once absorbed into the circulation. From this we have the chill, the high temperature, the prostration, and all the other evidences of a virulent infection. The rapidity with which the toxic product finds its way into the system is explained by the favorable conditions presented by the great vascularity of the lung and the enormous surface from which absorption takes place. Indeed, it would almost seem as if the lung structure were arranged with a view to such a result.

So long as fresh supplies of toxin are being formed, or, in other words, so long as the consolidation is spreading, so long the toxæmia will be maintained. But here again, as in artificial cultures, there is a limit beyond which the process cannot extend. A given quantity of a culture medium can maintain the life of a given number of germs only for a certain time. Beyond that time the changes produced in the medium render it unfit as a soil for the further growth of the organisms, and the death of the latter puts an end to the process. With the supply of toxin cut off the temperature falls. If the invasion has been regular and rapid, coming to an abrupt termination with the complete consolidation of the lobe, the supply of the toxin will cease abruptly, and we shall have defervescence by crisis. If,

on the other hand, the effusion into the air cells has been gradual, and the process of consolidation slowly carried forward, the supply of toxin will continue in one part while it fails in another; the process will be prolonged, and the defervescence will be by lysis.

This does not exclude the theory of the production of an antitoxin. Indeed numerous observations, and especially those of the Klemperer Brothers (see page 124) go to show that a transient immunity is created by an attack of pneumonia, and that this immunity may be transferred to another subject by serum inoculation. This could scarcely be explained except on the theory of an antitoxin, and I can see no obstacle to accepting both theories in explanation of the clinical phenomenon in question.

As bearing upon the formation of an antitoxin the following observation by Pinna[56] is of interest: He found that the pus obtained by injecting turpentine into the cellular tissue of a man, and which pus was absolutely sterile, had the power when injected into rabbits of rendering the animals immune to inoculations of pus containing pneumococci. The rabbits recovered and remained healthy, while others not so protected succumbed to pneumococcus septicæmia within thirty-six hours after receiving a like amount of pneumonic pus. It would seem from this that the pus itself, apart from any microbic action, possesses antitoxic properties.

This suggests the probability that the exudate during the process of retrogression assumes the character of simple sterile pus in its relation to the pneumotoxin. The theory of an antitoxin will be further discussed in treating of serum therapy.

But the matter is still further complicated by a chemical factor that enters into the case the moment the current in the functional capillaries is arrested. Normally there is carried in these vessels a varying amount of sodium bicarbonate held in solution in the blood. It is requisite that the carbon dioxide contained in the bicarbonate should be set free in the lungs, in order that it may escape with the expired air. Nature provides for this by the production of a special organic acid in the parenchyma of the lung, known as pneumic acid. This combines with the sodium, and the carbon dioxide thus liberated passes into the alveoli.

But when the functional circulation ceases, the sodium bicarbonate is no longer at hand to neutralize the pneumic acid, which continues to be produced as a part of the nutritive process. The parenchyma of the lung, therefore, becomes saturated with a free acid, and the slightest trace of acid in the medium is, as has been proved from laboratory experiments, fatal to the propagation of the pneumococcus.*

* The acid reaction of hepatized lung has been demonstrated in the cadaver.

Thus there are three things that tend to bring about the crisis: failure of the supply of toxin, the formation of an antitoxin, and the presence of a free acid in the affected part inhibiting the action of the diplococcus.

Probably the phagocytosis of Metchnikoff also plays a part, the phagocytes destroying the pneumococci. This, however, is only conjectural.

With regard to the mode of access of the germ to the air cell there are only three channels by which it might reach the seat of its activity in the lung. These are the blood-vessels, the lymphatics, and the air passages. As to the first of these it has been amply demonstrated that the pneumococcus may find its way into the blood-vessels, and may set up a peculiar action in localities that are shut out from all communication with the exterior of the body. Thus it is sometimes found in the fluid of pericarditis, and in the pus of meningitis, the lung not being at all implicated. We must therefore concede the possibility of its reaching the lung in this way; but is this the usual route? Against this supposition is the fact that in the vast majority of cases of pneumonia the local infection is strictly limited to the lungs, although, as we have seen above, the organism is capable of attacking other structures. Moreover, if we were to admit a selective action on the part of the lungs whereby the coccus is attracted, as it were, to the pulmonary structure (as the gonococcus is, for example, to certain tissues), we still have to account for the fact that as a rule the morbid process begins in a single focus in the lung and spreads from that as a centre, instead of attacking different places in the lungs, as it would be likely to do if the organisms were floating in the blood. When we add to this what the researches of Kanthack and others prove, that if in a case of pneumonia the pneumococci are found in the blood, and can be cultivated from it, the prognosis is bad, while, as we know, under usual conditions a large proportion of cases of pneumonia terminate favorably, we have a strong probability, to say the least, that the blood-vessels are not the usual channel by which the infection reaches the lung.

As to the lymphatics, the same reasoning holds good, and in addition we have the extreme improbability that the organisms could pass the barrier of the lymphatic glands.

This brings us back to the air passages as the most likely avenue of infection.* But this is by no means an open one. The anatomical

* As corroborative of this view of the mode of access of the infecting germ it is interesting to observe that as in two-thirds of the cases foreign bodies passing down the trachea find their way into the *right* bronchus, owing to its greater width, so we find in practice a very considerable preponderance of right-side pneumonia,

configuration of the bronchial tree is such that it is impossible that a solid particle, however minute, should be carried with the inspired air beyond the second or third divisions without coming into contact with the bronchial wall. Even gaseous material cannot pass from the glottis to the alveoli at a single inhalation. It is only by a series of inspiratory impulses, aided at last by molecular interchange between the inspired and the residual air, that it can reach the air cells.

Once deposited upon the mucous surface of the tubes all the conditions favor the expulsion of a solid particle rather than its further progress inward. In respiration the outward current is much more rapid than the inward, and expiration tends therefore to drive backward with a greater force than inspiration exerts in drawing forward. To this constant outward impulse is added the action of the ciliated epithelium, by which a movement in the direction toward the glottis is imparted to the fluid which bathes the bronchial mucous membrane. These influences combined render it extremely difficult for any solid substance, however minutely divided, to be carried into the alveoli by simple inhalation.

This provision of nature for preventing access of foreign material to the air cells is so complete that they are very seldom invaded by even the finest particles that are carried in the atmosphere.

Doubtless this provision is operative to a very great extent in protecting the lungs from the ingress of pathogenic germs. These may appear in the mouth and pharynx, where the epithelium is of the pavement variety, but when they reach the point where the ciliated epithelium begins, the action of the cilia opposes their further progress. But for this we should find all manner of germs disseminated over the whole extent of the respiratory tract, instead of being confined, as a rule, to the regions above the glottis and to the larger bronchial tubes.

But this protection is not absolute at the best, and is liable to be impaired by any cause that affects the bronchial mucous membrane. Thus, for example, the efficiency of the ciliary movement is lessened during the early dry stage of a common cold, and in bronchial catarrh.

Even if the protection remained intact it would not prevent the spread of microorganisms over the bronchial mucous surface by the process of multiplication, which process acts with inconceivable rapidity under favorable conditions. A single microbe lodged in one of the smaller tubes, and there finding a suitable culture medium in the secreted mucus, would in a few hours produce millions of its

probably the result of the same anatomical condition by which germs on reaching the bifurcation in their passage downwards are influenced to pass into the right bronchus rather than the left.

kind. The colony spreading in this manner would not be long in reaching the vesicles. Here the epithelium is of a different character, resembling closely the type of serous membranes, which are prone, under irritation, to yield a fibrinous exudate. In the alveolus, however, this form of exudate appears from present indications to be the product of only one form of irritation, viz., that resulting from the presence of microorganisms. It seems to be afforded principally, if not wholly, by the *pulmonary*, as distinguished from the *bronchial*, capillaries. When there is infiltration into the septum from its *nutrient* vessels the infiltration is corpuscular and not fibrinous.

As has already been seen, the pneumococcus lanceolatus is present in the mouth and nasopharynx in perhaps the majority of healthy persons. Dr. Neumann's studies made at my instance show that in three out of sixteen autopsies in persons dying of other disease than pneumonia this organism was found in the smaller bronchi, say, of the fourth or fifth division. Why in such subjects they do not always penetrate into the air vesicles and excite pneumonia is a question of great interest, the answer to which is not at hand. That they do so penetrate under certain conditions we already know, but what the conditions are, and how they act, we cannot tell. In general terms, their presence in the air cells seems to be associated with depression of vitality, as if the protection against their entrance was sufficient so long as the vigor of the subject remained unimpaired, but failed when it was diminished. Starr[25] suggests that the pneumococcus becomes more virulent after exposure to cold.

It will be urged against this view of what takes place in the pneumonic lung that there is no necessary relation between the amount of lung involved and the degree of toxæmia. This is true as a simple proposition; but in this as in all other forms of toxic infection, different persons show remarkable differences in susceptibility to the poison, and at the same time the poison itself varies extremely in virulence.

Furthermore, accepting the theory of an antitoxin, the period at which the formation of this substance begins in a given case and the rate at which it progresses, become at once factors in determining the general condition of the patient at a given time.

But perhaps the most important consideration in this connection is that the area in which the toxin is being formed may greatly exceed that in which physical signs can be detected. The thinnest film of exudate will be a sufficient medium for the growth of microbes, and that under conditions most favorable for a rapid absorption of the poison they afford. So that while there may not be material enough in the bronchioles and alveoli to change the percussion note,

or to give rise to crepitus, it may still be the source of very active infection.

Keeping these points in view we may safely say that, other things being equal, the systemic impression will be closely proportioned to the extent of the local invasion.

Occasionally in the progress of a pneumonia, gangrene of a portion of the lung takes place. We may assume that in these cases a branch of the bronchial artery becomes involved in the process, thus shutting off the nutrient circulation from the corresponding portion of lung tissue. That this does not occur more frequently shows how passive is the rôle played by the pulmonary parenchyma.

A process of less intensity involving the nutrient vessels would account for the permanent change found in chronic pneumonia.

Suppuration with the formation of abscesses in the lung tissue is now and again observed. This is probably due to a mixed infection, in which pyogenic germs take part with the diplococcus and produce a more intense degree of local irritation involving the lung structure itself.

The evolution of new germs having ceased, and the system having recovered in a measure from the depression of the toxæmia, the process of the removal of the exudate from the air cells begins. This is accomplished chiefly by absorption, and is facilitated by the liquefaction of the fibrin and the granular disintegration of the corpuscular elements which characterize the stage of resolution.

In the course of a few days, if all goes well, the cell is entirely relieved of its contents. Its walls remain intact and ready for an immediate resumption of function.

The sum of the argument, finally, is that from its peculiar construction the lung is enabled to afford a field for bacterial culture and to supply a culture medium, and this without calling upon its own nutritive resources, or directly compromising its own tissues. It is the fact that in the lung there are empty spaces accessible to bacteria, and separated from an unlimited blood supply by only the thinnest and most permeable wall, that makes the phenomena of pneumonia possible. It is the further fact that the framework between these spaces has its own separate blood supply, apart from the vessels involved in the pneumonic process, that prevents a sweeping destruction of the lung tissue.

In no other organ in the body is such a mingling of structural health and diseased action conceivable, for in no other organ is the blood supply for nutrition and function separately provided for.*

* A somewhat analogous condition is met with in the heart, inasmuch as blood flows through its cavities for functional purposes, while its nutrition is supplied

The sequence of events taking place in an attack of pneumonia would seem to be as follows:

1. The occurrence of some cause of depression, either local or general, which favors the germination of pneumococci, already present in some one of the smaller tubes.

2. The formation of a colony that spreads until it reaches the group of air vesicles that are terminal to the tube in question.

3. The setting up of an irritation in these vesicles, causing a fibrinous exudation, an emigration of leucocytes, and a diapedesis of red cells from the functional capillaries.

4. The formation of a colony of pneumococci in the medium afforded by this exudate.

5. Arrest of the blood stream in the functional capillaries, followed by accumulation of free pneumic acid in the parenchyma of the affected area.

6. Overflow of exudate into neighboring lobules, starting the process in them also.

7. Arrest of germ growth by exhaustion of the medium and the accumulation of free acid in the tissue of the lung. Up to this time there has been a constant formation and absorption of toxin.

8. Retrogressive changes in the exudate preparatory to its removal by absorption.

9. Probably, in this latter process, formation of an antitoxic principle.

10. Entire removal of the exudate and restoration of the vesicle to its normal condition.

11. Resumption of the functional capillary circulation.

The physical signs of pneumonia being in a typical case so distinctive, we are liable to fall into the error of considering that the disease is not present until some one or more of these signs can be made out. But, regarding as the first step in the process the invasion of the air cells by the germ, pneumonia is present the moment the first lodgment is effected. Investigation has shown that the pneumococcus is most active in creating toxin at the earliest period of its life, and also that the toxin it produces is then most virulent. Hence, we can understand that an amount of exudate not yet sufficient to be detected by the physical signs would still be sufficient to afford a medium for the growth of cocci in adequate numbers to poison the system with their toxin. Until such poisoning reaches a point at which general symptoms are manifested, fever, chill, etc., there may

through the coronary vessels. It would be a legitimate, if gross caricature of the old idea of pneumonitis if one should find a heart with its cavities filled with fibrinous clots and should label the specimen *carditis*.

be nothing to indicate what is going on in the lung except perhaps prodromes, which may or may not arrest the attention of the patient. In other words, an important stage of progress may be reached before the disease is openly declared. This explains those cases of very sudden invasion, as, for example, one cited by Sturgis, in which a person started to cross London bridge from the Surrey side without pneumonia, but had pneumonia when he reached the City.

The conditions for rapid absorption of the poison are best when the air cell is but partly filled; later, when the cell is crowded with exudate, and the affected portion of lung is distended beyond its natural bulk, the pressure renders absorption less active. At the same time, as the germs grow older, the toxin becomes less virulent, and a time arrives when the fully hepatized portion ceases to be an unfavorable factor in the case except for the breathing surface it withdraws from use. On the contrary, it may be that the older germs now reverse their rôle and begin the production of an antitoxin. Were it not for the fresh growth constantly spreading about the circumference of the hepatized portion and extending in a wider circle than that indicated by the physical signs, the infection would soon come to a standstill and crisis would result. As it is, the crisis is deferred until the infecting are balanced by the disinfecting forces, the latter including the various emunctories by which the poison is discharged from the body.

Bacteriology.

The history of the pneumococcus is interesting in that the organism was known for some time before its connection with pneumonia was suspected. During this interval it received various designations, such as micrococcus Pasteuri, micrococcus of sputum septicæmia, bacillus septicus sputigenus, etc. Later, when its pathogenic relations had been established, it received more specific names—diplococcus pneumoniæ, pneumococcus capsulatus, pneumococcus lanceolatus, pneumococcus of Fraenkel, micrococcus pneumoniæ crouposæ.

In 1880 Sternberg discovered to his surprise that his saliva, he being in perfect health at the time, was fatal to rabbits if injected into the cellular tissue, death taking place within forty-eight hours. Cultures made from the bodies of these animals resulted in the production of bacilli of a kind not before recognized, and inoculation with which was uniformly fatal. Waiving his right to give his own name to the new organism, he called it the micrococcus Pasteuri. These

observations were followed by others made by different bacteriologists and with results confirming the discovery of Sternberg. Netter found by inoculation experiments in rabbits that the saliva of one hundred and sixty-five healthy individuals showed the presence of this micrococcus in fifteen per cent. of the number.

In 1883 Talamon demonstrated the presence of this organism in the sputum of pneumonia, and produced pneumonia in rabbits by injecting material containing the germs directly into the lungs through the chest wall.

In 1886 Weichselbaum published the results of investigations showing the presence of the diplococcus in the fibrinous exudate of croupous pneumonia in the proportion of ninety-four cases in one hundred and twenty-nine. Since then it has been generally accepted that croupous pneumonia is an infectious disease caused by the pneumococcus lanceolatus, though other organisms may be associated with the latter.

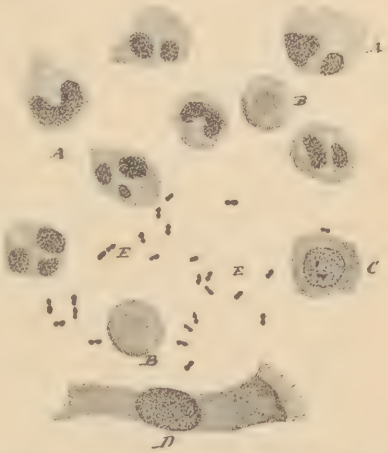

FIG. 3.—The Micrococcus Lanceolatus in Pneumonia Sputum. *AA*, Leucocytes; *BB*, red blood cells; *C*, epithelial cell; *D*, ciliated epithelium; *EE*, pneumococci. × 1000.

For the following description and the accompanying drawing I am indebted to Dr. George A. Tuttle, assistant pathologist of the Presbyterian Hospital, New York.

"The micrococcus lanceolatus, called also Fraenkel's diplococcus or pneumococcus, and diplococcus lanceolatus pneumoniæ, was first described by Sternberg in 1880 as the micrococcus Pasteuri. He noted its very frequent presence in normal saliva and demonstrated its virulence by animal inoculations. A. Fraenkel confirmed the observations of Sternberg, and noted the presence of this diplococcus in the rust-colored sputa of pneumonia, while it was absent in other acute inflammations of the lungs. He also obtained this organism, called by him the diplococcus of sputum septicæmia, in many cases of empyema following pneumonia and in the exudation of meningitis complicating pneumonia. In 1885-86 he was able to prove conclusively the causal relationship of this micrococcus to acute lobar pneumonia.

"The micrococcus lanceolatus in the blood of inoculated animals and in pneumonic sputum is generally seen in pairs of oval or lancet-shaped elements surrounded by a capsule of a substance resembling

mucin. In cultures short chains of three to four members are common, and at times longer chains of ten to twelve are seen, while the capsule is absent. There is a marked variation in the size and shape of the individual elements in cultivated specimens, and this peculiar characteristic is of considerable importance in distinguishing this organism from the streptococcus pyogenes which it so closely resembles in other respects. Like the other micrococci it is non-motile.

"It grows in all of the common faintly alkaline culture media, with or without the presence of free oxygen, at a temperaure of 35°-38° C. It will not grow at the ordinary room temperature, or on potato. On the surface of agar or of blood serum it forms minute, grayish, translucent colonies, at first resembling minute drops of water. In gelatin it grows rather slowly along the entire puncture and at the surface, the growth beneath the surface consisting of minute white colonies. It does not liquefy gelatin. It grows rapidly in milk, producing coagulation; also in bouillon, causing a slight cloudiness of the fluid and a little whitish sediment at the bottom of the tube. It loses its vitality in cultures on solid media in five to ten days; in bouillon its life is somewhat longer. By transplanting every fourth or fifth day the growth may be continued indefinitely in favorable conditions. The virulence of artificial cultures is rapidly lost, but can be restored by passing them through susceptible animals.

"It is readily stained with all of the aniline colors and by Gram's method. The capsule can be demonstrated in blood or sputum preparations by staining with carbol-fuchsin solution, and lightly washing with alcohol. The deeply stained diplococci will be seen surrounded by an oval colorless area representing the capsule."

This organism is found in the expectorated material in all but a very small percentage of the cases examined for it.* When appar-

*R. M. Pearce, in the Journal of the Boston Society of the Medical Sciences, for June, 1897, records that in 121 cases of acute lobar pneumonia the pneumococcus was found in the solidified lung in 110 cases, in 84 of which it was the only microorganism present. In the remaining cases it was associated with varying frequency with the staphylococcus pyogenes aureus and albus, streptococcus pyogenes, Klebs-Loeffler bacillus, and the bacillus capsulatus (Wright). In some cases the association was with two of the above. Of the remaining 11 cases, in 8 the lung culture was not taken in 4, lost in 2, and sterile in 2, but in each of these there was general infection with the pneumococcus, and in 3 it was present in either pericardial or pleural exudate. This would justify the conclusion that the process in the lung was due to the same organism and was the source of the general infection. If, therefore, these cases are added to the others they make a total of 118 out of 121, or 97.5 per cent. due to the pneumococcus. In the exudate of acute pleuritis and acute pericarditis with pneumonia, the pneumococcus was found in every case in which examinations were made, 49 times in pleural exudates and 15 times in pericardial exudates. In various infections due to the pneumococcus, numbering

ently absent it is probable that faulty methods of search have been employed, or that some special circumstance has masked its development. When a pure culture is injected into the substance of the lung it invariably results in a typical croupous pneumonia. Injected into the cellular tissue or into the peritoneum it causes septicæmia.

The coccus growing rapidly is commonly inert at the end of a week. Probably this contributes, at least, to determine the crisis. It may also explain the benign course of pneumococcic infection in other tissues. Such infection of the pleura, for example, results commonly in a mild and tractable empyema as contrasted with infection from other pathogenic organisms of slower growths and more enduring vitality.

The pneumococcus exhibits great differences in virulence, suggesting that there may be several varieties. Inoculation experiments appear to show that one of these, the œdematogenic, causes local œdema with toxæmia. This is the variety most commonly found in pneumonia. Another produces abscess at the point of inoculation, and "is an exquisitely pyogenic microorganism."[29]

Eyre and Washbourn[30] in a very elaborate paper demonstrate that "there are a large number of types of the pneumococcus which differ from one another in virulence and in biological characters." The parasitic type, the typical pneumococcus, is the most virulent and also the least hardy and has the shortest life. The saprophytic type, such as is found in the mouths of normal individuals, is almost destitute of virulence, but is very hardy and long lived. By repeated passages through animals the several types may be made identical as to virulence, but in the case of the saprophytic type this virulence is reached and maintained with difficulty. The maximum of virulence, obtained by eight passages of the parasitic type and fifty-three passages of the saprophytic type, was such that .0000005 mgm. injected into the peritoneal cavity of a rabbit was fatal within twenty-four hours.

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55 cases, there were 26 acute infectious processes which were neither preceded nor accompanied by a lobar pneumonia. The cases comprised acute ulcerative endocarditis, acute purulent meningitis, acute fibrinous pericarditis, acute fibrinous peritonitis, and acute general infection with pneumococcus.

James J. Curry, of Boston, in the Journal of the Boston Society of the Medical Sciences, for March, 1898, mentions two cases of acute lobar pneumonia in which the capsule bacillus (bacillus pneumoniæ of Friedländer) was found in cultures of the lung together with the diplococcus lanceolatus, the growth of the capsule bacillus being so powerful that it obscured entirely the development of the diplococcus. The results of bacteriological investigation of acute lobar pneumonia at the Boston City Hospital have shown it to be due invariably to the micrococcus lanceolatus.

When in a dry state the pneumococcus retains its virulence for long periods. This is especially the case when it is protected by being mixed with mucus in desiccated sputum. Cases are on record in which the disease was communicated to newcomers in houses that had been closed for many months. Schroeder refers to a house from which thirty-two cases came to the clinic at Kiel in the course of fifteen years, one year furnishing six.[31]

According to Simon,[32] recent researches show that in fatal cases of pneumonia the specific diplococcus is quite commonly present in the blood, while in cases ending in recovery it is only exceptionally encountered. Wirtz found positive results in eighty-nine per cent. of fatal cases.

In investigations conducted at the Presbyterian Hospital, New York, the blood was examined in twelve cases of pneumonia. Two of these ended fatally, the remaining ten in recovery. In none of them was a growth of the pneumococcus obtained in the tubes inoculated with the blood.[33] The invasion of the blood usually occurs from twenty-four to forty-eight hours before death, but may be earlier or later. For prognosis this may be important. Some cases end in recovery.

With a view to ascertaining whether there was any relation between the occurrence of pneumonia after operations under an anæsthetic and the presence of pneumococci in the upper air passages before the anæsthetic was given, the writer caused a culture to be made from the fauces of each patient anæsthetized for operation at the Presbyterian Hospital during a period of several weeks. The result was that it was found to be the rule rather than the exception that the coccus was present, and this being the case no conclusion could be arrived at on the point proposed for investigation. Indeed, it was only in a very small proportion of the cases that the cultures failed to develop the pneumococcus, not enough to determine whether its occasional absence offered even the slightest immunity from operation-pneumonia, itself an infrequent occurrence.

It was next sought to ascertain whether the organism existed in the deeper air passages with anything like the same frequency as above the glottis. At my request Dr. Neumann, in the laboratory of the Presbyterian Hospital, examined the smaller bronchi in sixteen cadavers of persons dead from other diseases than pneumonia. The examinations were conducted with great care, a number of cultures being made in each case from different localities in the lungs. The result showed the presence of Fraenkel's pneumococcus in four cases and the presence of an organism resembling Fraenkel's in one case, while in eleven cases no bacilli pneumoniæ were found.

The cultures were taken from the smallest branches into which the needle could be carried.

It would seem, therefore, that while the pneumococcus of Fraenkel is very generally present above the glottis, it occurs in only from one-fourth to one-third of the cases in the deeper portions of the respiratory tract. It is an interesting question what proportion of all the cases of pneumonia is to be found in subjects whose deeper air passages are already the habitat of the specific organism.

Diagnosis.

A typical case of croupous pneumonia seen from the beginning can hardly be mistaken for any other disease. The abrupt onset, the chill, the pain, the fever, the respiration accelerated out of proportion to the pulse and temperature, and, finally, the peculiar expectoration, will suffice to establish the diagnosis even without the aid of the physical signs. But when the latter are added, and we have fine crepitation with inspiration and a little later a blowing sound with expiration, while the vocal resonance and the vocal fremitus are exaggerated, and the percussion note becomes constantly duller until it approaches flatness, there is presented a clinical picture which for vividness and individuality can hardly be surpassed.

But cases are not always seen at the outset, or accompanied by reliable histories, and we may be called upon to make a diagnosis at any stage, and with little or no knowledge of what has gone before. This is often the case in hospitals, especially in those having an ambulance service, patients being brought in in a condition of delirium, or unable from natural stupidity or from ignorance of the language to give any account of their illness. Under such circumstances we must depend largely upon the physical signs, interpreting them by such objective symptoms as may be present at the moment.

If the signs are such as have been already indicated and the temperature is high and the breathing hurried, we may be sure that we have a pneumonia which has not yet defervesced.

If with the physical signs of consolidation we have a low temperature, and the breathing is but little accelerated, and there is more or less creamy expectoration, we may assume that the case is one of pneumonia which has passed the crisis. If under like conditions we detect crepitation in the consolidated portion, we have a commencing resolution signalized by the râle redux. Finally, if we have a temperature varying but little from the normal, slight percussion dulness, vesiculotubular breathing interspersed with coarse mucous râles, and accompanied by a mucopurulent and partly frothy expectoration, the

inference is that the case is one of pneumonia pretty well advanced in the process of resolution.

We must remember, also, that not all cases of pneumonia run a typical course. There is scarcely one of the classical symptoms or signs that may not sometimes be wanting. In about twenty per cent. of all cases the chill is absent. Pain is not a marked feature unless the pleura is involved, and in central pneumonia it is often not severe enough to excite complaint. In feeble or elderly persons the fever may be slight; indeed, even apart from these conditions, some of the worst cases we meet with show but a moderate temperature throughout. The pulse is likely to correspond with the temperature; and the respiration, usually the most characteristic of the symptoms, is sometimes not strikingly frequent. Cough and expectoration may be entirely absent through the whole course of the disease, or the cough may bring up only a little frothy mucus from the bronchial tubes.

As to the physical signs, all of them may be in abeyance for a considerable time when the consolidation is confined to the central portion of the lung. A greatly thickened pleura remaining from a former pleuritis may obscure the results of both auscultation and percussion, and lead to mistaken inferences.

Apart from the above considerations the diagnosis of pneumonia involves differentiation from quite a range of affections, such as bronchopneumonia, pulmonary œdema, pleurisy with effusion, abscess of the liver, pulmonary apoplexy, pulmonary phthisis, cancer of the lung, atelectasis, engorgement in fever, typhoid fever.

Bronchopneumonia is distinguished from acute lobar pneumonia by the less frequent occurrence of chill in the former, the less degree of pain, the pain being at the same time less fixed, a generally lower temperature, less increase in the rate of respiration as compared with temperature; while the cough is more severe, and the expectoration is more abundant, more frothy, less stained, and less adhesive. The disease is more frequently bilateral in the bronchial form than in the lobar. The dulness is much less circumscribed, and is more likely to appear in several places at once, and to shift its locality from day to day. The breathing over the affected part has not the tubular blowing type of lobar pneumonia, and is relatively feebler. Fine crepitation is absent, and in its place are subcrepitant râles heard with expiration as well as inspiration. The vocal resonance and vocal fremitus are less exaggerated. In short, the physical signs indicate that the consolidation is far less perfect than in the lobar form. As the case progresses we miss the distinctive crisis, and there are not the characteristic traits of a self-limited disease. In favorable

cases amendment may begin at any time, or it may be indefinitely deferred.

According to Delafield and Prudden,[1] "there is a form of bronchopneumonia in adults which resembles lobar pneumonia. There is a general catarrhal bronchitis, with bronchopneumonia and consolidation of one or more lobes. The symptoms and physical signs are those of lobar pneumonia, but with some difference. The invasion of the disease is not as sudden, the pulse is more rapid, the cerebral symptoms are more constant, the expectoration is like that of bronchitis, the physical signs are more slowly developed, the duration of the disease is rather longer and resolution is slower."

In pulmonary œdema we have cough, expectoration, embarrassed respiration, and some dulness on percussion. But there is no chill, no pain, no rise in temperature; the expectoration is profuse and watery, instead of small in amount and viscid. Pulmonary œdema is bilateral and begins usually in the most dependent portion of the lung, instead of being unilateral and occurring indifferently in one portion or another. In pneumonia we have the dry crepitant râle, while in œdema the râles are coarse and liquid. Bronchial breathing and bronchophony, heard in pneumonia, are absent in pulmonary œdema. Percussion dulness is less in œdema, and vocal fremitus is not exaggerated. The absence of chlorides from the urine so general in pneumonia is not observed in œdema of the lungs.

But it must be remembered that very many cases of pneumonia are complicated at some period in their course with pulmonary œdema, and we must always be alive to the possibility of the coexistence of the two affections.

Pleurisy with effusion has many points in common with lobar pneumonia. There is pain; there may be chill; there is fever; the respiration is hurried; there is a cough; and there is an area of percussion flatness. In some cases there is also bronchial breathing. When this is present the differential diagnosis may be far from easy.

But the fever is comparatively mild, the temperature rarely reaching 103° F., and the respiration does not fluctuate so widely with each change of temperature as is the case in pneumonia. There are not the nervous shock, the loss of muscular strength, the evidences of profound intoxication, seen in pronounced toxic cases of pneumonia. The cough is dry, there is but little expectoration, and what there is is frothy, not stained, not adhesive. If the effusion is considerable, the heart will be displaced towards the unaffected side.

Coming to the physical signs, we note the absence of crepitation, and generally, though not always, of bronchial breathing. On the

other hand, we have the presence in the early stage of a pleural friction sound, which is often enough found in pneumonia.

Percussion reveals an area of flatness (not dulness as in pneumonia) occupying what has been the most dependent portion of the pleural cavity on the affected side, with often increased resonance above. Sometimes the level of this area will change with change of the patient's position, but generally the fluid whose presence gives rise to the flatness is kept in place by interpleural adhesions. Over this area there is in most cases absence of respiratory murmur and of voice sounds, in place of bronchial breathing and bronchophony, though both of these are present in exceptional cases. Even in these cases there is absence of vocal fremitus, instead of the exaggeration observed in pneumonia.

There is one sign which if present and well marked goes far towards establishing the diagnosis, and that is ægophony above the line of flatness. There is nothing in pneumonia that fully answers to this, but it is not always present in pleurisy with effusion. The latter is true also in regard to bulging of the intercostal spaces and displacement of the heart or of the liver. Yet while there is no one sign or symptom which is always to be relied upon, unless it may be the absence of vocal fremitus, it is seldom that we need remain long in doubt, and in any case the matter may be cleared up by the harmless expedient of an exploratory puncture. We must bear in mind, however, that there are instances in which both conditions are present, and also that adhesions resulting from former inflammation may bring about some very perplexing distributions of fluid in subsequent attacks.

In simple hydrothorax there is an area of flatness with a somewhat bronchial character of the breathing and voice above it, but the upper line of the flatness, which is higher behind than in front, changes when the posture of the patient is changed. Ægophony is usually well marked. There is little or no fever, and the dyspnœa is comparatively slight, even when a large amount of fluid is present.

In pyothorax the physical conditions are much the same, and there are the symptoms of pyæmic infection, including wide excursions of temperature, sweating, etc.

In *abscess of the liver* and in subphrenic abscess, the pain and the rigor, together with an area of dulness, may suggest pneumonia of the right base. But the rigor is likely to recur again and again, there is absence of the auscultatory signs of pneumonia, and though cough may be present, we do not have the characteristic expectoration. If any doubt remains it is soon cleared up by the clinical phenomena, which, as the case proceeds, diverge widely from those observed in pneumonia.

In *pulmonary apoplexy* or hemorrhagic infarct we have pain often of a very severe character, frequent respiration, cough, an area of dulness, and, perhaps, bronchial breathing. These symptoms and signs suggest pneumonia, but the onset is too sudden, we miss the chill; the temperature is less elevated; the expectoration is abundant, and consists largely of pure blood. There are coarse râles within the lungs from the first. Moreover there is usually a history of antecedent cardiac disease, or of some infection by which the formation of pulmonary emboli or pulmonary thrombi is provoked. Attention to these points will suffice to establish the differential diagnosis.

Tuberculosis.—The consolidation attendant upon tuberculous deposit, especially if pleuritic pain be present, and if at the same time the temperature be considerable and the breathing frequent, might reasonably raise the question of the existence of pneumonia, if a history of the case should not be attainable. It might even be difficult if not impracticable at once to exclude this possibility. But the difference in the character of the sputa, the different temperature curve, and the afternoon hectic in phthisis would soon lead to a correct diagnosis. Besides, if in sufficient extent to raise the question of diagnosis, there would be likely to be auscultatory evidence of the breaking down of lung tissue. It is to be borne in mind, however, that an area of tuberculous deposit may be included in one of pneumonic exudation. If doubt remain, it may be removed by microscopical examination of the sputum.

Cancer of the lung has some features in common with pneumonia. There are pain, respiratory embarrassment, and cough, with expectoration which may be more or less stained with blood or otherwise colored. The physical signs of consolidation are also present in the affected region. But the onset is gradual and the disease protracted; there is little or no fever, no chill, the pain is continuous, there is a cancerous aspect after a time, and cancerous growth is apt to exist or to be developed in other organs. Indeed, there could be but little chance of confounding the two affections unless an inflammatory process should have been set up in a cancerous lung, and the previous history should not be obtainable, conditions which I met with once in consultation, and which had given rise to a diagnosis of unresolved pneumonia.

Atelectasis gives rise to circumscribed percussion dulness, and rapid respiration, but lacks the other features of pneumonia. Besides, it is a condition found almost exclusively in infancy.

Hypostatic Congestion.—During the course of a low fever, in which the vital powers are considerably depressed, there is likely to occur

a congestion of the lungs affecting especially the posterior portion. This gives rise to some shortness of breath, cough, frothy expectoration sometimes a little bloody, mucous râles, and some degree of dulness on percussion, and might be taken for the early stage of pneumonia. But its presence in both lungs, the absence of the crepitant râle, the comparatively slight effect upon the respiration, and the history of a preceding fever which still continues, render the distinction easy.

On the other hand, it often happens that in a protracted case of pneumonia symptoms arise that very closely resemble those of *typhoid fever;* and if the progress of the case has not been followed, it is quite easy to overlook the lung condition and see only the appearances that indicate a low form of fever. Indeed, not one of these may be wanting, and it may be only because we have more than is needed to constitute a case of typhoid that our suspicion of the true nature of the affection is aroused.

Of course, mistake would be possible only in the febrile stage of the pneumonia, and then the excessive frequency of the respiration should at once attract attention. In most cases the cough and expectoration would be significant; but granting these to be absent as they might be, there would still be enough in the respiration, including the unequal movement of the two sides of the chest, to show that the lung was implicated, and to lead to a physical examination which would be conclusive. How often this examination is neglected, however, may be inferred from the annual report of the Metropolitan Asylum Board for 1897, which states that among the cases of mistaken diagnosis are found sixty-one of pneumonia erroneously included in two hundred and ninety-three cases reported as typhoid fever.[34]

The routine application of the Widal test would reduce the chances of such error to a minimum.

The following is quoted from Osler[35]:

"Nervous symptoms are more frequent in pneumonia than in typhoid, and from the onset may so dominate that the local lesion is entirely overlooked. For instance, in the case of cerebral pneumonia of children, in which the disease sets in with a convulsion, there are high fever, delirium, great irritability, muscular tremor, and perhaps retraction of the head and neck, and consequently meningitis is usually diagnosed. Cases occur in which the malady sets in with acute mania. For example, a young man behaved so strangely on the train that he was handed over to the police as a lunatic, and as he had no cough and little fever (though he complained of a pain in the side) pneumonia was not recognized for several days. Again, pulmonary features are frequently marked where the patient has delirium tre-

mens, and error is certain to occur unless it is made an invariable rule to examine the chest in these cases. Then there are cases with toxic features resembling uræmia; without chill, cough, or pain in the side the patient may develop fever and a little shortness of breath and then gradually grow dull and heavy, and within three days there may be a condition of profound toxæmmia with low-muttering delirium. In many of these cases the most characteristic symptoms of the disease may be absent, particularly the cough and the rusty sputum; but the physical signs, if they are elicitable, are well marked. Even in the gravest of these cerebral cases the crisis and the onset of convalescence may occur in the ordinary way, and the patient may pass from a condition of extreme danger to one of perfect safety."

Complications.

Strictly speaking, the complications of pneumonia are only those morbid conditions which are so associated with the primary disease as to indicate that they are dependent upon it and would not have taken place in its absence. Conditions previously existing, or that are simply coincident, cannot rightly be considered as complicating the pneumonia, though they may be complicated by it. Thus an old cardiac or renal lesion can scarcely be said to complicate a pneumonia, though its presence may very materially affect the gravity of the case. On the other hand, an acute pericarditis or nephritis occurring in the course of a pneumonia, and evidently involved with it in some sort of causal relation, would be an illustration of what strictly constitutes a complication. There are several affections which so often appear with pneumonia as to make it reasonably certain that there is a common influence at work, and this influence can often be found in the presence of the pneumococcus in the locality in question. The following tables, compiled by Dr. Howland, show the complications observed at the Presbyterian Hospital, New York.

Complications of Pneumonia.

Out of 488 Cases.

	Number of cases.	Recovered.	Died.
Pleurisy with effusion	20	17	3
Pericarditis (acute)	6	1	5
Otitis media *	6	6	
Thrombosis of femoral vein	3	3	
Bronchitis †	13	9	4
Jaundice	2	1	1

* Otitis of catarrhal form in every case.

† These were cases complicated with well marked bronchitis from the outset and not those merely developing signs of œdema or of the softening of resolution.

Out of 304 Cases.

	Number of cases.	Recovered.	Died.
Gangrene of leg *	1	1	
Delayed resolution	7	7	
Relapse	2	2	
Delirium tremens	11	5	6
Pyæmia	2	..	2
Abscess of lung †	1	1	
Laryngitis	1	1	
Erysipelas	2	2	
Persistent bronchopneumonia	1	1	
Acute nephritis ‡	3	2	1
Œdema of lungs §	6	1	5
Empyema ‖	10		

Pneumonia Complicated by Coexisting Diseases or Conditions.

Out of 488 Cases.

	Number of cases.	Recovered.	Died.
Chronic endocarditis	11	3	8
Cirrhosis of liver	5	..	5
Fatty liver	4	3	1

Out of 304 Cases.

	Number of cases.	Recovered.	Died.
Chronic nephritis	7	1	6
Typhoid fever	1	..	1
Phthisis	4	..	4
Pregnancy	1	..	1
Sacculated aneurysm of aorta	1	..	1
Paralysis agitans	1	..	1
Chronic bronchitis	1	..	1
Influenza	6	6	
Cystitis	2	2	
Emphysema	2	1	1
Endarteritis	1	1	

* Caused by thrombosis of anterior tibial artery.

† Recovered after prolonged convalescence.

‡ These were well-marked cases without previous nephritis and with symptoms of acute nephritis much worse than transient albuminuria.

§ These are cases of œdema developing during the course of the disease.

‖ Satisfactory data could not be obtained as to the final outcome of these patients, but it might be interesting to notice the conclusion arrived at by Dr. Hartwell in a report covering ten years. Out of 52 cases of empyema treated in this hospital 26 gave good history of previous pneumonia and 6 more gave a possible history. Out of these 26 pneumonia cases 19 recovered and 7 died. He found that children with empyema, as a rule, did very badly unless treated early by operation; with such treatment then they did well.

Pleuritis.—The complication most frequently met with in pneumonia is pleuritis. Indeed the sharp pain, which is included in the familiar picture of the stage of invasion in pneumonia, is due, not to the disease of the lung itself, but to the involvement of the pleura. When the pneumonic process begins centrally, pain at first is either entirely absent or is manifested only as a dull ache, and it does not assume its characteristic severity until the disease has worked its way to the surface and taken hold upon the serous investment of the lung. In the great majority of cases, however, the pleura is very early involved, the stitch often being the first thing observed by the patient. Usually the pleurisy is dry, that is it gives rise only to a fibrinous exudate, which appears as well upon the parietal as upon the visceral layer, and ultimately glues the two surfaces together. In a minority of the cases there is a moderate amount of serous effusion which is later absorbed. Not very infrequently the pleura becomes the seat of pyogenic infection, and empyema results. This occurs in from one to three per cent. of all cases.* The infecting organism in these cases is the pneumococcus, which has worked its way from the lung into the pleura, and which in a serous or synovial membrane is prone to excite suppuration.

Usually the empyema occurs on the same side as the affected lung, and is then of moderate severity and runs a favorable course. But it has been observed that when this complication occurs on the side opposite the pneumonia, the prognosis is much more grave. This doubtless arises from the fact that in the first case the affection of the pleura is due to simple continuity of tissue, while in the second it is excited by cocci circulating in the blood. As already shown, cases in which the blood is infested with the organisms generally do badly, and it is to this circumstance, and not to any peculiarity of the pleuritis, that the gravity of the prognosis is to be referred. In such cases other organs than the pleura are extremely likely to be involved, either simultaneously or in succession, and under the pressure of multiplied foci of infection the patient speedily succumbs.

The friction sound of the pleuritis is quite likely to be mistaken for crepitus within the lung. Indeed the similarity of sound, especially to the subcrepitant râle, is very striking. But it cannot be gotten rid of, nor its character changed, by coughing; and this is a very important test. Of course, if the rubbing can be felt as well as heard this puts an end to all question.

Bronchitis.—Next to pleuritis bronchitis is the most frequent com-

* It occurred ten times in three hundred and four cases at the Presbyterian Hospital.

plication of pneumonia. In a considerable number of cases a bronchial catarrh precedes the pneumonic attack, and a common cold is said to have "run into pneumonia." The frequency with which persons are declared to have been "threatened with pneumonia" bears witness to the apprehension of such sequence which is rooted in the popular, not to say the professional, mind.

In a certain sense there is always a degree of bronchial catarrh in the affected portion of the lung. Invariably the mucous membrane of the tubes leading to a consolidated area is reddened by hyperæmia, and covered by a viscid mucus. Whether this is due to irritation by the sputum, or is a primary condition, in fact a part of the process deeper down in the lung, is a question to be considered. The view of the writer is that this hyperæmia marks the track down which the microbes marched to their proper field of activity in the parenchyma of the lung. Be this as it may, there is always evidence of such hyperæmia, and its products are mingled in greater or less proportion with the characteristic pneumonic sputum.

A general association of bronchitis with a form of pneumonia constitutes a disease by itself, bronchopneumonia, which has been already treated of by another writer in an earlier volume of this work.

Somewhat allied to bronchitis is the condition known as *collateral hyperæmia*, which is frequently developed in the course of pneumonia. It appears in the previously unaffected lung and consists of a more or less intense congestion. It is largely mechanical, and is the product of two factors. The first is the forcing into the sound lung of a portion of the blood which normally should have passed through the vessels of the obstructed lung. In this the right heart is the active agent. The second factor is the aspiration of an excess of blood into the sound lung by the action of the muscles of respiration. The inspiratory effort, not resulting in adequate expansion of the crippled lung, produces a condition of negative pressure in the sound one, and blood flows into the latter in consequence. This condition, however, is seldom serious except in advanced cases, when weakness and exhaustion of the right heart are a further cause for its supervention.

Bronchitis can be considered in the light of a complication of pneumonia only when in addition to the signs of consolidation over a given area there are bronchial râles scattered abundantly over both lungs.[2]

Gangrene of the Lung.—In rare instances a portion of the hepatized lung perishes, and a condition of gangrene is established. This is due to an involvement of the nutrient vessels in the process that

ordinarily interests only the functional blood supply. The precise mechanism by which the germ obtains access to a bronchial twig cannot be traced and probably is not the same in all cases. According to Osler,[23] "the gangrene is associated with the growth of the saprophytic bacteria on a soil made favorable by the presence of the pneumococcus or the streptococcus."

The extent of the gangrene will depend upon the size of the vessel at the point where infection takes place or to which the resulting thrombus extends in a backward direction.

The occurrence of this accident is marked by an intolerable fetor of the breath and in some cases by the appearance of the physical signs of a cavity. Occasionally the destruction of tissue opens a way into the pleura, and pneumothorax results.

Though a very grave complication gangrene is not by any means necessarily fatal. If the amount of lung involved be not too great, and the vitality of the patient not already too greatly depressed, a fair chance for recovery may remain.

Pericarditis.—This complication occurs in from two to eight per cent. of the cases of pneumonia, according to different authorities. It is often overlooked during life, the area of the pneumonic dulness including that of the pericardial effusion, and auscultation not being practised at the time when the pericardial friction sound might have been heard. At the Presbyterian Hospital it was made out during life in six out of four hundred and eighty-five cases of pneumonia. It is supposed to occur more frequently when the left lung is involved, and the inference is that the disease extends from the lung and pleura by continuity of tissue. More recent investigations, however, seem to show that there is no material difference in this respect. Doubtless it is more likely to be recognized during life when the pneumonia is on the opposite side, as the area of pericardial dulness in this case is not obscured by the dulness of consolidation.

It should be the rule in every case of pneumonia to examine the cardiac area at least daily. This is especially imperative when the respiratory and circulatory conditions appear out of proportion to the temperature and the nervous symptoms. Orthopnœa, in particular, suggests something more than the dyspnœa of pneumonia, and will often be found to be associated with effusion into the pericardium or the pleura.

The extreme flatness on percussion and the absence of vocal and respiratory sounds and of vocal fremitus over the effusion will generally suffice for the diagnosis. Auscultatory percussion may be of great value in fixing the limiting line between solid and fluid, a change in the character of the note occurring at once in passing from

one to the other, though to ordinary percussion there might be no appreciable difference.

When pericarditis occurs during the progress of pneumonia the pneumococcus lanceolatus may very generally be found in the effused fluid. It is frequently associated with other organisms, but under such conditions as leave little doubt that it is the determining factor.

A pericarditis occurring as a complication of pneumonia is much more likely to be purulent than when it is primary or associated with rheumatism. In such cases pneumococci may be found in the blood, and collections of pus may form in other serous or synovial cavities. Such conditions offer little hope of recovery.

Acute Endocarditis.—This affection appears in pneumonia both as an acute condition engrafted upon an old lesion, and as a fresh invasion apart from previous anatomical change. In some cases both the pneumonia and the heart affection seem to be the outcome of rheumatic poison.

It may assume the simple form with warty vegetations, or the malignant or ulcerative type with the pneumococcus as the active agent. In either case the effect of the disease is so intensified by the associated pulmonary affection that opportunity for a complete development of the clinical history is seldom afforded. This would be still more the case, were it not that the valvular trouble involves the left side of the heart, while it is the right side that has to bear the brunt of the struggle with the unfavorable pulmonary conditions.

The following case, seen in consultation with Dr. Janvrin, of this city, illustrates an acute pneumococcic infection engrafted upon an old valvular lesion:

Mr. T——, age 67, had had for years a valvular lesion, which, however, caused him no very serious inconvenience. On December 18th, 1898, after some two weeks' confinement to the house with influenza, which did not extend below the larynx, he had a severe chill, and signs of pneumonia soon developed in the right base. There was little cough and no expectoration. Chills recurred at irregular intervals thereafter, and not yielding to quinine and arsenic, the suspicion gained ground that they were of mycotic origin, which suspicion was soon confirmed by the occurrence of murmurs indicating a fresh endocarditis. Meanwhile the pneumonia cleared up very kindly, perhaps assisted by the massive doses of quinine. But the chills, perspiration, and extreme prostration persisted with increasing severity, and death took place on the seventeenth day of the attack.

Although an autopsy could not be obtained, there can be no doubt

that the organism which excited the endocarditis in this case was the pneumococcus. Drs. Janeway and Armstrong, who also saw the case, concurred in the diagnosis.

Chronic Endocarditis.—This is not properly a complication of pneumonia, but rather a preëxisting condition. At the Presbyterian Hospital, New York, of four hundred and eighty-eight cases of pneumonia eleven occurred in persons already having chronic valvular lesions. It is a very serious combination in view of the embarrassment of the circulation which it involves. Indeed, if the leakage is considerable, or the compensation defective, the chance of recovery is very small. Of the eleven cases above referred to eight proved fatal. A chronic valvular lesion is also important as affording a possible seat for an acute infective process, as in the case just cited.

Myocarditis as an actual complication of pneumonia can very seldom be substantiated. The high temperature to which cardiac degeneration is so often attributed is not often of sufficient duration in pneumonia to bring about this result. But pneumonia may attack a person who already has a softening of the cardiac walls, and in such a case the crippling of the heart adds greatly to the danger of heart failure or, at least, of hyperæmia and fatal engorgement of the lung.

In the absence of a previous history of recognized cardiac weakness it may not be easy to say whether a softening of the myocardium, found post mortem, existed before the pneumonia or was induced by it. But we may be aided in our judgment by the character of the heart beats in the early stage of the attack, as well as by the degree and duration of the temperature.

Parotitis occurs in a small proportion of cases of pneumonia either during the course of the disease or as a sequel to it. It is rarely seen in young or vigorous persons, but belongs to the later period of life, or to conditions of depressed vitality. In all probability it is the result of microbic invasion by way of the parotid duct. It nearly always eventuates in suppuration, and it resembles the parotid abscess which occurs in typhoid fever.

It is a very serious complication both because of its occurrence in enfeebled persons, and because of the somewhat formidable character of the affection itself. It more frequently affects only one of the glands, which fact points rather to a local than a general source of infection as above indicated.

Diarrhœa is occasionally met with in the course of pneumonia. In some cases it seems to be one of the results of the first shock of the infection. In others it occurs at a later period, and may be due to the resorption of the exudate, inasmuch as it continues after the

crisis. Doubtless the cause lies in many instances in excessive feeding under conditions very unfavorable to proper digestion. It is seldom a serious complication.

Jaundice is an infrequent complication. It was noted in two cases out of four hundred and eighty-eight at the Presbyterian Hospital, though other observers have met with it in a larger proportion of cases. The precise conditions upon which it depends have not yet been decided. It is not attended by the evidence of obstruction, and is probably the direct effect of the specific poison acting upon the liver. It is usually slight and of no practical significance. Occurring, however, in a patient already having a fatty or cirrhotic liver it is of more serious import. It is sometimes associated with gastroduodenitis.

Otitis Media.—This occurred six times in four hundred and eighty-eight cases at the Presbyterian Hospital. In all these cases it was of the catarrhal variety and ended in recovery. Purulent otitis media, however, is occasionally met with, and in these cases the pneumococcus may usually be demonstrated in the pus. It is fair to presume that the coccus finds its way into the middle ear through the Eustachian tube. The purulent form adds a considerable element of danger to the existing pneumonia.

Neuroses.—Among the neuroses complicating pneumonia Isager[36] observed aphasia in a boy nine years old. The aphasia was complete, the boy being unable to speak though understanding what was said. After a fortnight the aphasia gradually disappeared.

Leszynsky[37] presented a man before the New York Neurological Society suffering from neuritis of the brachial plexus which had come on in the course of pneumonia.

Voute, of Amsterdam,[38] observed external oculomotor paralysis arising in a case of apical pneumonia, the paralytic symptoms subsiding as resolution took place. He considered the paralysis to be of toxic origin rather than meningeal, as there was complete absence of cerebral symptoms.

Pregnancy.—The existence of advanced pregnancy adds greatly to the seriousness of an attack of pneumonia. The interference with respiration occasioned by the upward pressure is a matter of grave moment. In addition to this, premature labor is very likely to occur, imposing a heavy tax upon the already overburdened system. In the early months the danger is much less. Should the life of the fœtus be destroyed at this stage of the infection the abortion may be delayed sufficiently to give time for recovery from the pneumonia. An interesting case is reported by Verlet[39] in which a pregnant woman was attacked with pneumonia followed by a relapse. Premature de-

livery resulted, the child born at seven months was cared for in an incubator, and both mother and child did well.

Venous Thrombosis.—Thrombosis of the femoral vein is met with not very infrequently. Three cases are recorded in the four hundred and eighty-eight mentioned above. Increased fibrinosis seems to be a characteristic of the pneumonic state, and probably this condition lies at the bottom of the coagulation in the vessels. As in thrombosis from other causes, the left leg is more frequently affected than the right.

Da Costa[1] in a clinical lecture refers to three cases of this kind which have occurred in his practice. I saw a similar case in private practice some years ago. The vein remained occluded years afterward, and the limb became swollen after much standing or walking.

In the treatment of recent cases of thrombosis local frictions should be carefully avoided lest the clot be dislodged and carried into the circulation. Clots may be formed in the vessels of the interior, and doubtless many of the cases of sudden death observed in pneumonia are due to their translation into the pulmonary veins.

Meningitis.—Diplococci have been reported as found in the pus of meningitis by many bacteriologists, and these organisms have been frequently and fully identified as "diplococcus pneumoniæ." Netter, in 1889, in a report of 25 cases of purulent meningitis, states that 13 of these were examined microscopically, by cultures, and by inoculations into animals, 6 microscopically and by experiments on animals, and the rest microscopically only. Six of the cases were complicated with pneumonia, 4 with purulent otitis, and 3 with ulcerative endocarditis. In 16 of the 25 cases the pneumococcus was found; in 4 the streptococcus pyogenes, in 2 the diplococcus intracellularis meningitidis of Weichselbaum, in 1 Friedländer's bacillus, in 1 Newman and Schaffer's motile bacillus, and in 1 a small curved bacillus. Out of 45 cases gathered by Netter, this micrococcus was found in 27; streptococcus pyogenes in 6; and the diplococcus intracellularis meningitidis of Weichselbaum in 10.

In 4 cases of cerebrospinal meningitis, reported in 1889, Monti demonstrated the presence of this micrococcus in 3 of which pneumonia was a complication. In 2 cases the staphylococcus pyogenes aureus was present with the diplococcus pneumoniæ. Weichselbaum, in 29 cases of ulcerative endocarditis examined in 1888, found the diplococcus pneumoniæ in 7.

Testi in 1889, in a case of parotitis complicating lobar pneumonia, obtained the micrococcus pneumoniæ crouposæ from the pus in pure cultures, and in another case of pneumonia complicated with a purulent pleuritis, bilateral parotid abscess, and multiple subcuta-

neous abscesses, the pus from these varied sources all contained the diplococcus in large numbers. This was shown by microscopical examination and by inoculation into rabbits. Gabbi (1889) obtained the same coccus in pure cultures in a case of tonsillitis resulting in abscess.

In a considerable number of cases of otitis media this micrococcus has been found, and in pus secured by paracentesis of the tympanum, and frequently in pure cultures; by Zaufal (1889) in 6 cases, Levy and Schraeder (1889) in 3 out of 10 cases in which paracentesis was performed; by Netter (1889) in 5 out of 18 cases occurring in children. In 1889 Monti and Belfanti both reported cases of arthritis of the wrist-joint complicating pneumonia, in which this micrococcus was found in pure cultures. Ortmann and Samter in the same year obtained the diplococcus pneumoniæ in pure cultures in a case of purulent inflammation at the shoulder-joint following pneumonia and pleurisy.[12]

Prognosis.

The prognosis in pneumonia is modified by a number of pre-existing conditions, the principal of which are sex, age, season of the year, habit as to the use of alcohol, and the presence or absence of certain chronic diseases.

It is conceded that while pneumonia occurs more frequently in men, it is more fatal in women. Of 223 patients with pneumonia admitted into the Presbyterian Hospital, 170 were males, with a mortality of 28.8 per cent., and 53 females, with a mortality of 31.2 per cent.

PNEUMONIA—434 CASES.

Relation to Age.	Died.	Percentage dying.	Recovered.	Percentage recovered.
Below 5 years	0	0	13	100
Between 5 and 10 years	0	0	18	100
" 10 " 15 "	1	9	11	91
" 15 " 20 "	7	23+	23	76+
" 20 " 30 "	28	22+	95	77+
" 30 " 40 "	37	37+	62	62+
" 40 " 50 "	31	42+	42	57+
" 50 " 60 "	16	47+	18	52+
" 60 " 70 "	16	66+	8	33+
Over 70* years	5	62+	3	37+
	141	32+	293	67+

*It is not to be inferred, however, that the increased mortality as age advances is due entirely to senility. The greater frequency of preëxisting morbid conditions comes into the reckoning. The man of sixty has had two chances to contract bad

Before the age of two years lobar pneumonia is a rather rare disease. After that age the mortality is greater in proportion as the patient is older. The preceding table, based upon four hundred and thirty-four cases treated at the Presbyterian Hospital, New York, shows not only the frequency of occurrence, but also the mortality at the different ages.

Statistics accumulated by Frankel and Reiche (Maragliano") covering 1,130 cases give the mortality by ages as follows:

From	1	to	5 years	30.0	per cent.
"	6	"	10 "	3.84	"
"	11	"	20 "	10.05	"
"	21	"	30 "	8.70	"
"	31	"	40 "	24.70	"
"	41	"	50 "	39.30	"
"	51	"	60 "	43.10	"
"	61	"	70 "	63.60	"
"	71	"	80 "	86.70	"

The relative mortality of pneumonia in the different seasons of the year is variously stated by different writers. In the above two hundred and twenty-three cases, the spring months gave the lowest mortality, the summer showed a slight increase, while in the autumn and winter the death rate attained its highest percentage.

The habitual abuse of alcohol unfits the system to bear up against pneumonia. Of 11 such cases, referred to by Ewing as occurring at Roosevelt Hospital, only 3 recovered. The records of the Presbyterian Hospital covering 428 cases give the following results as to mortality among those markedly alcoholic, those moderately alcoholic, and those non-alcoholic.

PNEUMONIA—428 CASES.

Relation to Alcoholism.	Died.	Percentage dying.	Recovered.	Percentage recovered.
Markedly alcoholic*	36	70	15	29
Moderately alcoholic	52	32	109	67
Non-alcoholic	45	20	171	79

A preëxisting rheumatic habit has generally been observed to add to the gravity of the prognosis. It was present in 20 of the 223 Presbyterian Hospital cases and was attended by a mortality of 40 per cent. As the rheumatic habit, however, is more pronounced as

kidneys for one chance that has come to the man of thirty; and so through all the list of chronic diseases whose presence renders an attack of pneumonia more perilous.

* A noticeable fact was that of the fifteen recoveries, most of them were in young men between twenty and thirty years.

life advances, the influence of age in these cases must not be overlooked.

The prognosis is extremely unfavorable in diabetic patients. The combustion of the sugar, already imperfect, is diminished in proportion to the impairment of the respiration. Death in such cases is preceded by delirium and coma.

The preëxistence of chronic nephritis makes the prognosis of pneumonia more grave in proportion to the renal insufficiency and the accompanying cardiovascular changes.

Pneumonia occurring in a subject of advanced cardiac disease is likely to prove fatal. The prognosis is less grave before secondary changes have taken place in the right side of the heart.

Coming now to the conditions arising in the course of the disease, and which seem to influence the mortality, the first is the initial chill. This occurred in 144 out of the 223 cases, with a mortality of 34 per cent., while the remaining 79 cases in which the chill was absent gave a death rate of only 19 per cent. From this it is evident that the chill is an expression of a severer grade of infection, and I have observed that usually a prolonged rigor with tardy reaction presages an unusual degree of muscular and nervous prostration.

The prognosis depends largely upon the extent of the pneumonia. When both lungs are involved not half of the patients recover. Pneumonia occupying the whole of a lung is more dangerous than when only a part of the lung is involved.

As to the location of the lesion, the right lung is more frequently affected than the left, and also gives a higher mortality. Of 94 cases implicating the right lung alone, 28 ended fatally, giving a mortality of 29.8 per cent., while only 62 cases involved the left lung alone, of which 16, or 24.5 per cent., were fatal. In 39 cases both lungs were involved, with a mortality of 19, or 48.7 per cent. Previous attacks do not affect the prognosis.

A feeble pulse that is frequent in relation to the respiration and temperature has much the same prognostic significance as a heavy and prolonged chill. In my experience it indicates a profound specific infection, that is likely to carry off the patient at or before the crisis.

The respiration is always frequent in pneumonia, but when extreme frequency is associated with a high temperature, it has less significance than when it occurs without this association. In the former case it is due in part to the fever; in the later it denotes extensive lung implication, and probably œdema, always a perilous condition.

Up to 105 the danger does not seem to increase materially with

the rise of temperature. Thus in 45 cases with the maximum temperature ranging between 100° and 103° the mortality was 26.6 per cent., while in 99 cases with temperature between 103° and 105° the mortality was 26.3 per cent. The conditions that raise the temperature above 105°, however, immediately tell upon the death rate. In 49 cases with a temperature between 105° and 106° the mortality was 32.1 per cent., and 13 cases at 106° and upwards gave 61.5 per cent. of deaths. Recovery, however, may take place though the temperature attain an extraordinary height. Wagner has reported such a case in a boy five years old, the temperature having reached 109.2° F. Indeed, it is an error to attach too much importance to the temperature alone, so long as the condition otherwise remains favorable. Measures intended merely to control the pyrexia are indicated only in extreme cases. It cannot be too often repeated that to give coal-tar preparations according to indications furnished by the thermometer is to expose the patient to a real danger in an effort to rescue him from a fancied one.

There is a physical sign to which I called attention some years ago that I regard as important in the prognosis of pneumonia, and to which I have already referred in another connection. This is the character of the valve sound heard in the pulmonary area. The resistance in the pulmonary circulation which always exists in these cases must necessarily increase the tension in the pulmonary artery, provided the force of the ventricular systole is not impaired. With this increased tension come a sharper recoil of the blood against the valve cusps and a greater intensity of the valve sound. It follows, other things being equal, that in this intensity we have practically a measure of the degree of obstruction to the blood current in the lungs.

It is generally very easy to note as consolidation progresses the constantly increasing sharpness of the pulmonary second sound, and in favorable cases this accentuation, after reaching a certain point, is maintained with little change until commencing resolution renders the circulation easier, when the valve sound loses in intensity in proportion as the pulmonary condition improves.

But, unhappily, loss of accentuation of the pulmonary second sound does not always depend upon relief of the pulmonary circulation. It may signify instead a weakening of the right ventricle and a lessened power to force the blood into the artery. This comes when the right heart begins to flag either from exhaustion from excessive labor, or because of the depression arising from the specific infection. In either case, when we find the pulmonary second sound becoming weaker without any evidence of a favorable change in the condition

of the lung, we may draw the conclusion that the right heart is failing, and that the tendency of the case is towards a fatal termination.

The information furnished by the pulmonary valve sound is much more reliable than that supplied by the radial pulse, for in most cases the chief peril is of failure of the right heart from exhaustion, and of this the condition of the radial pulse will give only indirect and inconclusive evidence. The right ventricle may be on the verge of exhaustion, while the left retains almost undiminished vigor, and the radial pulse then will simply indicate that less blood than usual is flowing beneath the finger, as the result of paucity of blood in the left ventricle.

Unfortunately it will sometimes happen that the region of the pulmonary area is occupied by noisy râles, so that it may be difficult or perhaps impossible to appreciate the action of the valve. But unless the dyspnœa is extreme the breath can be held for an instant, and for the practised ear the briefest moment is sufficient. An emphysematous condition of the left apex may also obscure the sound of the valve or render it inaudible.

As to the prognostic value of the data furnished in this way, I can only say that I have never seen disaster come in a case of pneumonia unless it was preceded by a period in which the pulmonary valve sound was progressively losing in intensity. Or, to put it in other words, I have never seen a case with sharp and distinct pulmonary valve sound in which a favorable prognosis as to the immediate future was not justified by the event. The weakness of the right heart that is indicated when the valve sound becomes less intense takes place chiefly in two classes of cases. The first is that in which the system is overwhelmed by the virulence of the infection. These cases are marked by a severe and protracted initial chill, great muscular prostration, a small and relatively frequent pulse, and marked involvement of the nervous system. We may well believe that in such cases the heart muscle is poisoned, and that consequently the right ventricle with the increased labor thrown upon it early becomes exhausted. Such cases are apt to prove fatal before defervescence takes place. If they can be carried beyond this period the prognosis is greatly bettered.

The second class of cases is made up of those in which from age, intemperance, previous illness, or other cause, the integrity of the circulatory apparatus is compromised in advance. Here we have a constant tendency to the invasion of fresh areas of lung, and to the supervention of œdema. This tendency, while it increases the labor required of the ventricle, is itself increased in proportion as the ventricle weakens under its burden.

The exact relation between the temperature and leucocytosis in pneumonia is not yet fully determined; but in general terms it may be stated that a high temperature in a vigorous subject carries with it a large number of leucocytes. From a prognostic point of view this is of great importance. Whether we consider the leucocytes as scavengers, and assume that they destroy the cocci which are the medium of infection, or whether we assign to the white cells a share in the production of antitoxin, certain it is that severe cases in which there is marked leucocytosis do better, as a rule, than similar cases in which the increase of white cells is relatively slight.

When the leucocytosis is inconsiderable it will generally be found that the system is not reacting energetically against the infection, a condition which suggests an unfavorable prognosis.

On the other hand, there may be a high degree of leucocytosis which is not due to the pneumonia pure and simple, but to some complication as pleuritis or pericarditis, and if this is not taken into account a faulty prognosis may result.

Ewing, in an excellent article on this subject,[41a] gives the count of the white cells in 101 cases, of which 37 were fatal. He says: "An examination of the deaths will show that in severe forms of lobar pneumonia a slight leucocytosis is a very unfavorable sign. In 6 fatal cases the number of leucocytes was subnormal. In 11 cases the average number was 9,000. Not one case recovered, in which the disease was even of moderate severity, when the number of leucocytes fell below 14,000. In several instances, again, a slight leucocytosis seemed at the time the only unfavorable prognostic sign in cases ending fatally."

M. Laehr,[41b] in an article on "Leucocytosis in Pneumonia," published in 1893, maintains that in pneumonia there is an unmistakable connection between the temperature and the leucocytosis. When the disease is at its acme the count of the white cells is at the highest, while at the crisis there is a falling-off. In pseudo-crisis and deferred resolution the leucocytosis continues though the temperature falls. In the generally constant agreement of the course of the fever, the amount of the lung infiltration, and the leucocytosis, the author sees a dependence upon a fourth factor, viz., the intensity of the infection, that is, the quantity and quality of the bacillus poison and the power of reaction of the individual. He goes on to say: "Still more important is the prognostic significance. If the number of leucocytes does not fall with the decline of the fever we may conclude that the process has not yet arrived at a standstill. If it falls with the fall of the temperature, but rises again the following day, we must be pre-

pared for a renewed attack of the fever or the occurrence of some form of complication."

R. C. Cabot[12] gives the count of the white cells in 49 cases of pneumonia. Leucocytosis was present in 41 cases with 10 deaths; absent in 5 cases with 5 deaths; doubtful in 3 cases with 2 deaths. He says: "So far as these figures go, therefore, it would seem that while the presence of leucocytosis is not a very hopeful sign, its absence makes the outlook bad. Moreover, in two of these five cases the other reasons for giving a bad or guarded prognosis were not present. The individuals were both under fifty, were not at all alcoholic or weakly, and there was nothing in the condition of the pulse, temperature, or physical signs in the chest to lead one to expect a fatal result. Yet both went down with surprising rapidity, and without any reaction to vigorous stimulation and supportive treatment."

In a subsequent paper[13] he adds twenty-four new cases, without materially changing the conclusions to which his figures would point.

Regarding this subject Dyce Duckworth[14] concludes that both clinical inquiry and laboratory experiments have shown that leucocytosis is usually a favorable sign in pneumonia. This is well established by the observations of von Limbeck, Billings, and others who find that an active inflammatory leucocytosis during fever usually indicates a good reaction and while not invariably so, is often a good prognostic sign. The cells increased in number are the multinuclear or neutrophile, or finely granular eosinophile cells. Conversely, absence of leucocytosis is, in any case, usually a bad omen. Kanthack and Lloyd declare diminished leucocytosis with falling temperature to be a favorable sign, while a continuous high temperature with a small number of leucocytes is unfavorable. They also noticed that a persisting or increasing leucocytosis with fever is favorable. A satisfactory prognosis is not established by the presence of leucocytosis, but it is a most valuable indication when taken with associated temperature, the curves of which are often roughly parallel. Kanthack and Romer confirm these views through experiments upon animals, since they found that on injecting pneumonic bacterial toxin, if recovery took place, there was at first a sudden decrease in the number of leucocytes, followed by a rapid increase together with fever, and again persisting leucocytosis, with a gradual disappearance of the excess of leucocytes as the temperature fell. When a fatal dose was injected, the leucocytes diminished in number, and continued so, while the temperature became subnormal.

A remarkably high leucocytosis, especially if associated with a low temperature, suggests a suppurative complication. Thus in a case of pneumonia at the Presbyterian Hospital the temperature of

the seventh day was 103°, and the count 54,500. The crisis occurred the next day, but with a temperature of only 99° F. there was still a leucocytosis of 42,000. Examination showed that an empyema was developing. In another case with a similar complication the count was 44,000, while the temperature was 101.3°. In eight uncomplicated cases the highest in eighty-one daily counts was 41,000.

At the Presbyterian Hospital, in cases in which we wish to follow the leucocytosis from day to day, we have adopted the plan of recording it on the temperature chart, allowing for each additional 3,000 leucocytes a space on the chart equal to one degree of temperature. Regarding 9,000 as the normal, this figure would correspond with the normal temperature line; 13,000 would be recorded on the 100° line; 16,000 on the 101° line; 19,000 on the next line above; and so on, the tracing being done in red ink (in the accompanying chart [No. 3, page 28] in dotted line.) In this way the relation of the count to the temperature is apparent at a glance.

Much has been said regarding the prognostic significance of herpes labialis, an affection that occurs very frequently in the course of pneumonia. The general opinion is that it is of good augury, though why it should be so is not easily explained. Tallman,[16] however, concludes that it is due to individual peculiarity, many persons having outbreaks at other times and, therefore, being liable to an attack during pneumonia. It is not critical and may appear early or late indifferently. It is not coincident with a fall of temperature or a change in the general or local conditions. Its prognostic significance is generally, though not always, good. Germain Sée observed a mortality of 9 per cent. in cases with herpes as against a mortality of 25 to 30 per cent. in general.

Dyce Duckworth[17] considers that it is a good sign and usually accompanies cases in which a well-defined crisis occurs on the sixth day. Cases that are grave and those that are prolonged with deferred crisis, usually do not present it. He says further that a good sign, even in a lean and elderly patient, is an abundant rusty sputum with a fresh bright color. But when the sputa are of a "green-gage" color or bistre tint the augury is not good, and sputa of a dirty orange color are of distinctly unfavorable prognosis. The prognosis in Bright's disease is grave and indications of dextrocardiac distention and general cardiac failure are invariably serious. Favorable symptoms at the crisis are sweating and some diarrhœa.

According to Loomis,[1] the appearance of prune-juice expectoration is an unfavorable symptom, as it indicates a depraved condition of the blood. If there is an absence of expectoration in the second and third stages or if the expectoration becomes scanty and difficult

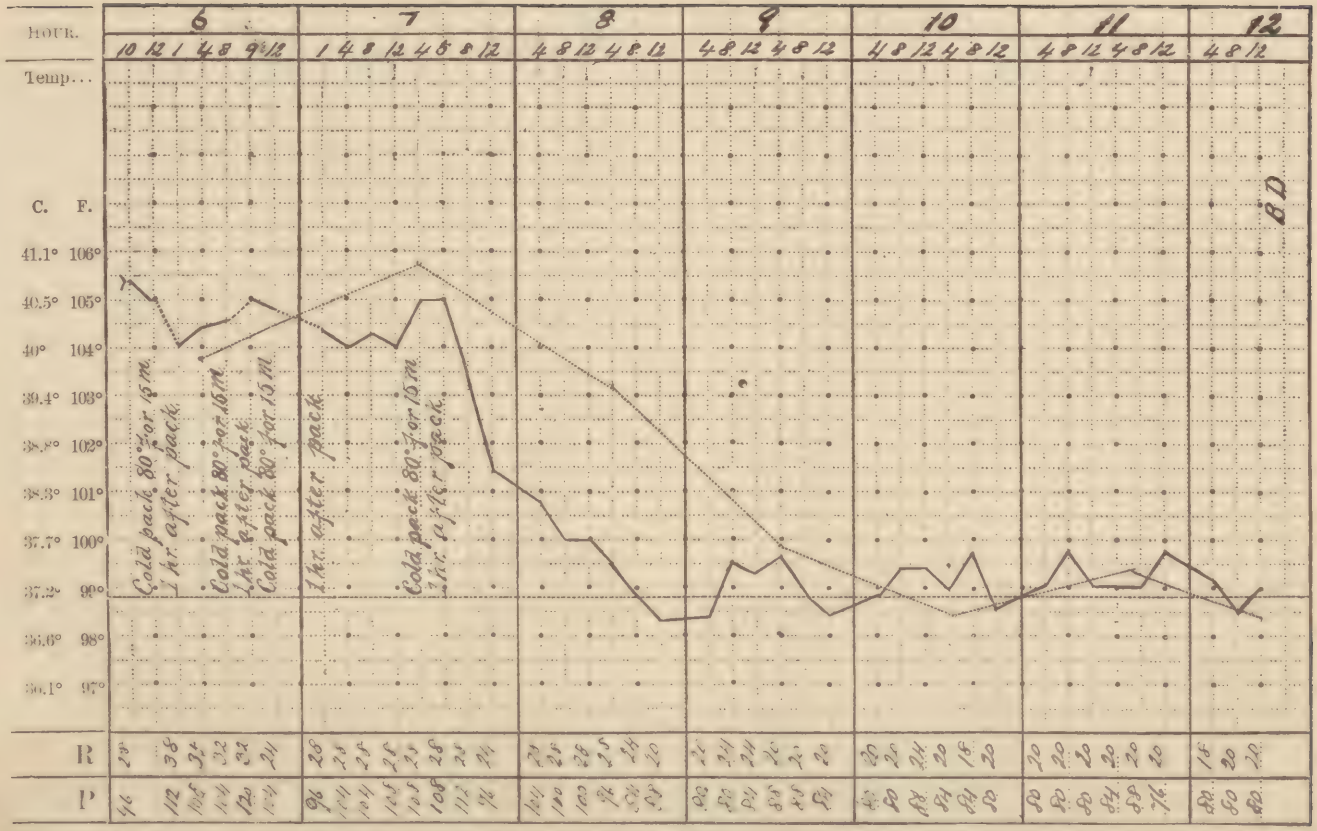

CHART No. 3.—The patient entered hospital on the sixth day of the disease with a temperature of 105.5°; highest leucocytosis on the seventh day, 29,000; normal temperature by crisis on the eighth day; normal leucocytosis on the tenth day.

the outlook is grave. Any sudden cessation of expectoration at any stage of the disease, if accompanied by tracheal râles, indicates the near approach of death. Delirium is an unfavorable sign except when it occurs early in the disease.

In the aged a sudden rise or fall of temperature, apathy, somnolence, and a sallow complexion, are all indicative of great danger.

The mortality in pneumonia varies enormously under different conditions. It is much greater in hospitals than in private practice, and in civil than in military hospitals. In civil hospitals the patients are usually taken from a class of persons imperfectly nourished, under bad hygienic conditions, and often with impaired constitutions as the result of these conditions. Then, too, only the very worst cases are sent to hospitals, the milder ones being treated at home. Add to this that the hospital patients as a rule do not present themselves until the disease is far advanced, and that they are subjected to the exhaustion due to removal, and it is not remarkable that the death rate in such institutions is very high. Of the last 458 cases treated at the Presbyterian Hospital 154 terminated fatally, being about 33.2 per cent.

Osler[68] gives the mortality in eight large hospitals as follows: Montreal General Hospital, 20.4 per cent.; Massachusetts General Hospital, 25 per cent.; Charité New Orleans Hospital, 38 per cent.; Boston City Hospital, 29.1 per cent.; Pennsylvania Hospital, 29.1 per cent.; St. Thomas', London, 20 per cent.; St. Bartholomew's, London, 18.6 per cent.; Edinburgh Royal Infirmary, 28.8 per cent.

This gives an average of 26.1 per cent., which pretty fairly represents the result of hospital practice. The greater mortality at the Presbyterian Hospital is due to the ambulance service, which brings in many patients who are practically moribund on admission.

In military hospitals, where the patients are young, vigorous males, and are admitted at the first moment of the attack, the mortality ranges from 3½ to 7½ per cent.

In private practice the conditions are for the most part the reverse of those obtaining in civil hospitals, and the mortality is proportionately less. For obvious reasons it is not practicable to accumulate accurate statistics, but the general fact is patent to every practitioner.

There has been for the last few years a very general impression in the minds of the profession that the death rate in pneumonia in this country had rather increased than diminished during the last two or three decades. Statistics gathered by Dr. Coolidge, of Boston, seem, however, to refute this idea.

Epidemics of pneumonia differ greatly in the death rate which

attends them. The same is true of different years and different localities, even when no epidemic influence appears to be prevailing. There are epidemics in which a large number of persons are affected, but the mortality is small. In other epidemics "almost every person is doomed" (Wells[1]). In certain years nearly all the cases in a given locality terminate favorably, while in other years in the same locality the mortality is very great.

These facts can scarcely be explained except on the assumption that there is a difference in the microbe at different times and in different places, similar to the differences existing in the cultures obtained in the laboratory.

These variations in the type of the disease probably afford the explanation of the phenomenal success of given forms of treatment in the hands of certain practitioners, and their utter failure when employed at other times, and in other places, and by other practitioners. They should warn us not to accept too readily the enthusiastic praises of particular methods (backed up it may be by an array of favorable statistics) which are passing in constant procession through the medical periodicals. It has happened to most of us, no doubt, to have had a succession of favorable cases under a particular management, until we came to believe that at last we had learned how to treat pneumonia. But just as we were beginning to rest in this assurance, we have been chagrined by encountering an equal array of failures with our trusted method, and have been ready to conclude that all treatment was of doubtful value. The lesson to be learned is, that an intelligent application of principles is to be aimed at, rather than a search for some particular method or formula.

Etiology.

While the essential cause of pneumonia is the development of a specific germ in the pulmonary alveoli, it is evident that as a rule there must be a contributing cause that in some way lays the system open to attack. We know that the pneumococcus is present in the upper respiratory tract in very many persons in perfect health, nay more, that it may be found in the blood, and even may be the pyogenic agent in a meningitis, a pleuritis, or a synovitis, and yet the lung escape. We must therefore assume that in any given case there is a condition present that favors either the migration of the germ into the lung or its development when there, or both.

Some of the causes that bring about this condition we are able to recognize, though we may not be able to trace their mode of action.

Sex seems to be a predisposing cause, since males are more fre-

quently attacked than females. That this is not due wholly to difference of occupation and habits, but partly to difference of structure or of constitution, is shown by the fact that in infancy boys are more frequently attacked than girls. Men are more exposed than women to cold and dampness and to excessive and exhausting labor, all of which increase the liability to pneumonia. Intemperance, too, is more common among men and adds greatly to the risk.

The relative frequency in the two sexes is variously stated; Wells,[15] from an analysis of 33,606 cases, gives 23,853 as occurring in males, and 9,753 in females. In 223 cases at the Presbyterian Hospital, in New York, the figures were 170 and 53 respectively.

Age.—Considering the total number of persons living at any given age, it is probable that the ratio of cases in successive decades is fairly uniform, except during the most active period of life, when exposure is greatest. Senility can scarcely be said to increase the liability to attack, though it adds immensely to the danger if an attack occurs.

Race.—In America, the negro race seems to be decidedly more liable to pneumonia than the white. This is clearly shown by the statistics of the Southern States, and also by the reports of army medical officers in the charge of colored troops. In the latter case it cannot be due to difference in mode of life, as the conditions are absolutely the same for all soldiers irrespective of color. It is doubtless only a manifestation of the peculiar delicacy of the respiratory organs characteristic of the negro when removed from the original habitat of his race.

Former Attacks.—Unquestionably a person who has passed through one attack is more liable to incur another. Instances are cited in which as many as eight or ten or even more attacks have been recovered from. In such cases it is probable that a bacterial residuum remains from one seizure to the next.

Unsanitary Living.—Pneumonia occurs more frequently in crowded, dark, and ill-ventilated dwellings than where the supply of light and air is ample. The life of the tenement house is peculiarly favorable to its development, and the crowded, stuffy, overheated workshop is responsible for an undue proportion of cases. Fortunately the germ is short-lived, or the disease would become endemic in such locations. As it is, the conditions render the lungs especially vulnerable to the action of the specific organism and increase the chances of infection.

Such being the principal predisposing causes of pneumonia, we have now to consider those which appear directly to determine the attack.

The first, and by far the most frequent, is *exposure to cold*. The connection between such exposure and the subsequent attack is often too direct to leave any doubt of the causal relation. For example, of persons rescued after falling into very cold water a considerable proportion will have pneumonia, and that without having been submerged or having taken water into the air passages. But even comparatively slight chilling of the surface, especially if continued for a considerable time, is very frequently followed by an attack which may, or may not, be preceded by the usual symptoms of a cold. A period of unusually low temperature very generally leads to an increased prevalence of pneumonia, especially among young children and aged people. The former for the most part get well, but after a long cold snap there is pretty certain to be a notable increase in the obituary notices of old people, most of whom will have died of the disease in question. How it is that this impression of cold brings about the specific disease in the lung we do not really know. It may be that the temporary arrest of the function of the skin causes a retention of excrementitious matter in the blood, which, seeking elimination through the lungs, determines there a local irritation favorable to the development of the germ. Or the constringing effect of cold upon the cutaneous vessels, by forcing the blood back upon the internal viscera, may induce such a congestion of the pulmonary capillaries as to favor the initiation of a pathological process. A chilling of the surface is very generally attended by a sense of discomfort in the lungs which warns us that we are in danger of "taking cold."

To this may be added a dryness of the bronchial mucous membrane, which, by impairing the action of the cilia, deprives the deeper portion of the lung of the protection that the ciliary movement affords against the invasion of harmful particles, the pneumococcus included. And, lastly, it may be that cold occasions a general lowering of the system, making it an easier prey to morbific influences. This last is the explanation usually given, but it is difficult to understand how such a degree of depression should be so quickly induced, and why it should not manifest itself in other ways as well, and also why much greater depression produced by other causes does not have pneumonia as a result.

While in a large proportion of hospital cases the disease is found to have followed upon a debauch, we are not warranted in assuming that intemperance as such is a cause of pneumonia. But it so often leads to exposure, and so generally interferes with regular and proper living, that its victims are always the subjects of impaired vitality, and therefore especially liable to this disease.

While it is popularly believed that a common acute bronhiale

catarrh may "run into pneumonia," there is no clinical evidence of such a tendency. When pneumonia occurs it is very rarely preceded by bronchitis or other pulmonary affection, and the presence of such a condition does not expose to greater risk of pneumonia. Even tuberculous subjects are not for that reason more likely to contract the affection, and it is just as likely to seize upon another portion of the lung as upon the seat of tuberculous deposit. On the other hand, pulmonary emphysema and asthma seem to confer some degree of immunity against pneumonic attacks. Flint thinks that this is true also of valvular disease of the heart.[55]

Other infectious diseases seem often to open the way to infection by the pneumococcus. The specific fevers, typhus and typhoid, measles, erysipelas, dysentery, each is a frequent forerunner of pneumonia, and holds a causal relation to it. In these cases the pneumonia is modified by the preëxisting disease, and seldom follows the regular clinical course. It is apt to assume a wandering form, appearing in patches in different parts of the lungs, presenting irregular and fluctuating temperatures, lacking a definite crisis, etc.

The morbidity varies considerably with the different *seasons* of the year. According to Flint,[47] the period of greatest frequency is during the spring months, the winter months come next, while the period from June 1st to December 1st affords the smallest number. This corresponds with observations at the Presbyterian Hospital in New York.

In reference to *climate*, pneumonia occurs more frequently in the southern portion of this country than in the Northern States, where the air is colder, and the conditions are apparently more favorable for its development. Thus it is evident that it is not a question of temperature, but of some other influence, the nature of which is not yet understood, but which probably has a relation to the life history of the specific microbe.

The frequency of the disease at the same season of the year and in the same locality varies greatly in different years.

Pneumonia seems to be more prevalent in high altitudes than at places nearer the sea-level. The epidemics of pneumonia occurring in Switzerland are mostly confined to the high valleys, while the reverse is true with regard to epidemic catarrh.[48]

Arthur Trower[49] mentions a case of septic pneumonia in a patient who had suffered from spinal paralysis for three years and was in feeble health. The sepsis apparently resulted from the pressure of, and inattention to, a dental plate that had not been removed for several weeks. Upon its removal a gangrenous condition of the mucous membrane of the hard palate was discovered, and the continuous

inhalation, swallowing, and absorption of the poison was the undoubted cause of the pneumonia.

Kronig[50] insists that it is dangerous to place a patient with pneumonia near one suffering from tuberculosis, as the latter infection is peculiarly liable to engraft itself upon the former.

Post-Operative Pneumonia.—The use of anæsthetics by inhalation in surgical operations seems to be occasionally the cause of pneumonia. Silk quotes Cheevers as giving pneumonia a prominent place among the causes of death after operations. This was in 1843, before the days of anæsthesia. In Cheevers' paper are given some observations of Erichsen, showing that of forty-one deaths after surgical injury no less than twenty-three subjects presented signs of pneumonia. In contrast to this, modern surgery, as shown by Silk presents only one case in 5,000 operations.[51] Prescott, on the other hand, estimates that in 40,000 cases of etherization there were only 3 cases of acute lobar pneumonia.[52]

At the Presbyterian Hospital, New York, in the ten years from 1887 to 1897, there were 4,914 administrations of ether followed by 17 pneumonias, 9 of which were fatal; 689 administrations of chloroform with 8 pneumonias, 7 fatal; 116 administrations of ether and chloroform with 2 pneumonias, both fatal.

It will be seen from these figures that pneumonia after anæsthesia is much oftener fatal than under other conditions, and that the administration of chloroform is much more likely to be followed by a fatal attack than when ether is employed. Schultze, however, from whose report the above statistics are taken,[53] does not believe that this large mortality following chloroform inhalation is due to the anæsthetic chosen so much as to the fact of the great number of cases of malignant disease of the mouth or respiratory tract among those to whom chloroform was given.

Of the cases at the Presbyterian Hospital 18 occurred in males and 9 in females. The right lung was involved in 14 cases, the left in 6 cases, both in 3 cases; no record in 4 cases. Pain was one of the earliest symptoms, and was felt often a day or two before the temperature rose noticeably or physical signs could be made out. Only 5 of the 27 cases were ushered in by a chill, and of these 5, 3 were fatal. The rarity of the initial chill in these cases is worthy of note, also the fact that the absence of chill did not indicate, as it usually does, a milder course of the disease.

Pneumonia after operations seems to be much more frequent in England and Germany than in this country. In the clinic at Erlangen during the period 1887 to 1894 there were 38 ether narcoses with 6 pneumonias, of which 4 were fatal. During the same period there

were 300 narcoses with chloroform with 15 pneumonias, 4 fatal. In abdominal operations the use of ether seems to be followed by pneumonia with especial frequency.

Whitney[1] suggests that the mouth should be disinfected before taking the anæsthetic, to destroy the specific germ and prevent its descending into the lung. Lucas has raised the question whether the frequent occurrence of pneumonia after operations may not sometimes be due to infection from the inhaler and suggests measures for avoiding the danger.[2] Bad ether may contribute to the result in some cases.

When we consider, however, the great frequency of pneumonia after operations before the days of anæsthetics, it is evident that their use prevents vastly more cases than it causes, if indeed it is ever the active agent in the production of the disease. It seems much more fair to say of ether and chloroform that they sometimes fail to protect the patient from post-operative pneumonia, than that they ever bring about an attack. This protection comes probably through lessening of shock and of nervous depression. It is possible also that the anæsthetic may exert a direct antagonistic influence upon the coccus.

Communicability of Pneumonia.

While pneumonia is unquestionably a communicable disease, it is not readily transmitted from person to person. From what has been already stated in regard to the very frequent presence of the specific germ in the air passages of healthy persons, it will be inferred that something more is required than simply the transfer of microbes from one individual to another in order to communicate the disease. Nevertheless, there is much in literature to show the occurrence of limited local epidemics of pneumonia, as well as the appearance of a number of cases in single households, either simultaneously or in rapid succession. In addition to this, certain houses have been observed to furnish year after year an undue proportion of cases, apparently indicating that the infecting principle lurked in the apartments.

During the building of the new Croton aqueduct in New York a large number of Italian laborers were employed. They congregated together in wretched huts along the line of the aqueduct, sleeping in a confined and contaminated atmosphere, and living in ignorance of every principle of health. Pneumonia was of very frequent occurrence among them. In 1886 Darlington[3] treated one hundred and fifty cases. The conditions were such as to invite pneumonia apart

from transmission from one person to another, yet doubtless this contributed to the result.

In Middleborough, England, there occurred in one year (1888) 367 cases of pneumonia in a population of 40,000. Halwell reports the occurrence of 50 cases in 13 days in a village of 400 inhabitants.

Emmerich[57] reports an epidemic of pneumonia occurring in a prison at Amberg in 1880, and lasting for six months, in which 161 cases occurred and 46 proved fatal. In dust found beneath the flooring of the dormitory in which the cases were treated, Emmerich demonstrated the presence of the pneumococcus, and obtained results similar to Friedländer's by inoculating animals with it. Dust from the floor of other dormitories in which there had been no cases of pneumonia was examined without detecting pneumococci. After thorough cleaning and disinfection no further cases of the disease appeared.

Tyson[59] says: "Out of a ship's crew of 815, 410 have been attacked in rapid succession, and of 720 attacked 298 fell victims."

In hospitals, and under ordinary conditions in private families, there is, however, very little danger of communication. The apparent instances of such transmission are probably quite as often due to depressing causes incident to grave illness in the household, or to prolonged and exhausting attendance at the bedside. In my experience I have seen but a single instance in which I thought the disease was directly communicated, and this was in the person of a hospital nurse, who while in attendance upon a patient with pneumonia was himself fatally attacked.

As, however, it has been shown by Flügge and others that in the act of coughing a fine spray may be formed from the expectoration, which spray is capable of floating in the air for an hour or more before it subsides, it is quite possible that the inhalation of this may convey the specific germs directly into the deeper air passages and thus excite an attack. An instance of communication apparently in this way was narrated by Girdner in a discussion before the New York Academy of Medicine, December 17th, 1896. In this case the nurse, who was very constant and faithful in her duties, lifted the patient's head at each attack of coughing, and held the towel into which he expectorated. She developed fatal pneumonia during the progress of the case.

The many similar instances in literature leave little room to doubt that the disease is communicable under very favorable conditions, and it is therefore incumbent upon the medical attendant in each case to adopt precautions against such infection.

As to the etiology of pneumonia in general, we are obliged to

admit that in a large proportion of cases the attack seems to come on spontaneously, that is to say, there is no appreciable antecedent condition which appears to hold a causal relation to it.

Treatment.

The first step in the treatment of pneumonia is to place the patient in a single bed, so situated as to be accessible from either side. Space also should be left for passing between the head of the bed and the wall. The room should be large and well ventilated and if practicable should be lighted by a window on each side of the head of the bed, and not by one at the foot. The temperature should be kept at about 65° F., the attendants wearing a little extra clothing if necessary, especially for the feet. The air of the room should be completely changed every two or three hours according to the size of the apartment, and in the intervals a window should be kept slightly open, the bed being protected by a screen if required.

The modern high bedstead now generally adopted in hospitals adds greatly to the comfort of the physician and attendants. In view of the probable use of water more or less freely for sponging, etc., the mattress should be protected by rubber cloth.

The patient should be clothed in a soft flannel gown which should be made to open in front to facilitate examinations, local applications, etc. The bed clothes should be light and not so abundant as to bundle up the patient too much or to retain the exhalations from the body.

Clean cloths should be provided for receiving the expectoration, and these should be burned before they have an opportunity to dry. The sheets should be changed frequently and "antiseptic precautions" observed in every particular.

Alimentation.—Undoubtedly the usual tendency is to give too much food to pneumonic patients. The extreme adynamia sometimes seen in the course of the disease, together with the oft-repeated injunction of lecturers and writers "to keep up the strength," frequently results in as much nourishment being crowded upon the patient as he can be induced to swallow. But the febrile condition is unfavorable to digestion, and fermentation with consequent flatulence often results. The abdominal distention in turn interferes with the descent of the diaphragm and adds to the difficulty of respiration.

All this is the result of a mistaken zeal in the matter of feeding. The fact is overlooked that the loss of strength is due to the intensity of the infection which poisons nerve and muscle, and not to lack of nutriment. Furthermore, even if the food were digested and absorbed into the circulation, there would remain the equally impor-

tant function of assimilation to be accomplished before the system could profit by the nutritive material added to the blood. Assimilation depends largely upon oxidation, and when the respiration is seriously embarassed hæmatosis is correspondingly imperfect. Under these conditions an excess of pabulum, even if taken into the blood current, can only add to the burden of the system.

The condition of the alimentary canal, therefore, should be constantly watched, and only so much food and of such a quality should be given as will be completely digested and assimilated. The appearance of stomach or intestinal flatulence, or of a thick and muddy urine, should lead to a readjustment of the diet. This may consist at first chiefly of milk, of which forty ounces per diem will usually give a better result than a larger quantity. If not readily digested the milk may be peptonized, and if it acquires a bitter taste by the process being continued too long, this may be covered by the addition of a little coffee. Kumyss or matzoon may be substituted wholly or in part, if more agreeable to the patient.

Beef tea, or expressed beef juice, may be used alternately with the milk to relieve the monotony of the diet.

There are several food preparations on the market that supply both nitrogenous and starchy elements already acted upon by their appropriate digestive ferments. These preparations contain some alcohol, added to preserve them, and they therefore serve both as food and stimulant. Such products, coming from reputable laboratories, may be relied upon as to their composition, and can be made to fill an important place in supplementing other articles of diet. They are not likely to cause flatuence, and are generally acceptable to the patient.

During the pyrexia, whatever food is given should be in liquid form and in small quantities at short intervals. After the crisis, if the digestion be fairly good, the diet may be more generous; eggs, fish, and finally meat being added as the appetite returns.

Throughout the disease the patient should have as much water as he desires. An active diuresis tends to carry off the toxins in the blood, and the best diuretic is pure water in abundance.

Treatment of the Local Condition.

When we have pneumonia we have an area of lung in which every air cell represents a tiny culture tube filled with a culture medium, and harboring a colony of diplococci; the lung tissue meantime being nourished by a separate set of nutrient vessels which are not materially involved in the process. This is the essential condition; incidentally more or less breathing surface is withdrawn from use,

and the system is flooded with a toxin more or less virulent. Unhappily what is incidental in the situation is of much more serious import than what is essential.

Now it is clear that this state of things is absolutely unique. We have not, and for anatomical reasons we cannot have, anything resembling it in any other locality. Analogy, therefore, can aid but little if at all in the matter of treatment. Nor can therapeutical methods based upon former pathological conceptions be utilized otherwise than as they may furnish authenticated facts having a value apart from their theoretical setting. Hence, there would be little practical utility in entering upon the history of the treatment of pneumonia. Until very recently, except so far as it was wholly empirical, this has merely reflected the current views as to the proper management of inflammation in general. The local process having been regarded as inflammatory, the disease has shared in turn the onslaught of the lancet, of mercury, of tartar emetic, and of all the long list of antiphlogistics. Even up to the present moment we hear of the use of certain drugs or methods to "reduce the inflammation" at the same time that the infectious nature of the disease is recognized. With this recognition, however, many, if not most authorities show a disposition toward therapeutic nihilism. "The disease is infectious and self-limited, there is nothing for us to do but to guide it if possible to a favorable termination." But the generalization that would classify pneumonia with scarlet fever and smallpox simply because it is technically infectious, is almost as unfortunate as that which would label it an inflammation and give it nosological fellowship with everything that ends in *itis*.

Keeping before us the nature of the disease, it is evident that a rational treatment must have both a local and a general bearing. The local bearing will require in the first place measures tending to lessen the production of toxic material in the lung, and in the second place measures that obviate so far as may be the mechanical obstacles to respiration. The general bearing will be in the direction of minimizing the toxic effects upon the system at large, or enabling the system to bear up under these effects.

Over and above the indications thus afforded, special conditions which may arise will require to be met by special measures.

Moving upon these lines the first object is so to act upon the blood as to make the fibrin which is exuded from it into the air cells as unfit a medium as possible for the growth of the specific germ.

In considering the possibility of thus restraining the activity of the local process we come at once upon two facts important for our encouragement. The first is that the life of the diplococcus is very

short, not exceeding ten or twelve days at the most in artificial cultures. The second is that of all known germs this is perhaps the most sensitive to its environment, laboratory experience showing that it can be cultivated successfully only by the most careful attention to its habits and peculiarities. The slightest deviation from the conditions these impose puts an end to its growth. Furthermore, the probability of successfully inhibiting the action of a germ through the influence of an agent diffused in the blood is greatly enhanced if the germ is located in the lung. This is due to the fact that the whole mass of the blood passes through the comparatively small pulmonary circulation every time that it traverses the vastly greater systemic circuit. Hence any substance in the blood comes into much more intimate contact with a germ in the lung than it would with a germ placed elsewhere, and the assault is proportionately concentrated and energetic.

It is true that to be fully effective a germicide designed to act within the air cell must be employed before the circulation in the functional capillaries is arrested. After such arrest it can reach its destination only by the very narrow channel of the nutritive blood supply. But as the pressure of the exudate is the ultimate factor in closing the functional vessels (whatever coagulating influence the morbid process may have exerted) it is not until consolidation is complete that access through these vessels is entirely cut off. It is also encouraging to note, in looking back, that various methods have been found useful in a marked degree, and have even been credited with the power of cutting short the disease in a certain proportion of cases. These measures were employed either before germs were known, or certainly without any germicidal intent, but we now feel that they owed their value chiefly to their antimicrobic influence.

Mercury.—Foremost among these is the use of mercury. Half a century ago this was universal, and it is hard to believe that intelligent and acute clinicians, men, for example, who could draw such an admirable picture of pneumonia as that furnished by Sir Thomas Watson, could have been wholly deceived in their estimate of the value of this agent. Watson[60] says (the italics are mine): "Many persons I am persuaded are saved by treatment of this kind, pushed to slight ptyalism: *the effusion of lymph*, tending to spoil the texture of the lung, *is arrested, and the lymph already effused begins to be absorbed:* and the ease and comfort of the patient, *as well as the alteration for the better of the physical signs*, attest the healing qualities of the remedy."

Twenty-five years later, Dr. James R. Leaming[61] writes: "I well remember my astonishment when thirty years ago the late Dr. E. P.

Cammann ordered a large dose of calomel in an attack of intercurrent pneumonia in a case of chronic phthisis, and my gratification at seeing the disease successfully controlled thereby. It was perhaps the most practical of all the valuable lessons I received from him. . . . The admirable *sedative effect* of calomel when needed is best seen when it is placed dry upon the tongue of the patient. . . . *The temperature at once begins to fall* (italics mine), *the heart to gain strength*," etc. . . .

Personally I have witnessed too often both in Dr. Leaming's practice and my own the beneficial effect in pneumonia of a single dose of thirty to fifty grains of calomel, given early, not to share to some extent in the enthusiasm of the writer.

The New York *Medical Journal* for June and July, 1879, contains a very interesting report on the "sedative" dose of calomel, made to the Therapeutical Society of New York by Dr. Putnam Jacobi, Secretary of the Committee on Antipyretics. It is a summary of the results of a collective investigation covering fifty cases in which large doses (twenty to sixty grains) of calomel were given. In most cases the object was to reduce an inflammatory process. In nearly all there was a marked decline of temperature within twelve to twenty-four hours after the dose. Fourteen cases were of croupous pneumonia, all of them severe. Of these patients thirteen recovered and one died. This is a remarkable showing, and it is impossible to read the report and not yield to the conviction that in some of these cases the calomel turned the scale toward recovery. In no case was there excessive purging from so large a dose. Stomatitis occurred only twice in the fifty cases, and produced no serious inconvenience.

At the present time we can scarcely accord to mercury the "antiphlogistic" property with which it was formerly credited, and Leaming does not ascribe the benefit derived from it to such a quality but to its *sedative action* when given in the (large) *sedative dose*. We can understand, however, that when an agent so inimical to germ growth is taken into the blood and effused into the air cells along with the fibrin that is to act as a culture medium it must have an effect to retard at least the activity of germ formation in that medium. Laboratory experiments show that the pneumococcus is peculiarly sensitive to mercury; and with so large a quantity of calomel diffused through the alimentary canal it is easy to conceive that enough mercury, probably in the form of the bichloride, should be absorbed and exuded with the fibrin to sensibly affect the pabulum upon which the microbe feeds. *A fortiori* would this be the result when the blood is so charged with mercury as to produce the phenomena of ptyalism.

More recently, Pieragnoli[62] strongly recommended the employment

of calomel in croupous pneumonia. His method consisted in the daily administration of calomel combined with opium and in the avoidance of expectorants in the first few days of the attack. His results were very satisfactory. Of five patients to whom the calomel was not given all died, of fifteen who were treated with calomel all but one recovered. Equally good was the result in children. The course of the disease was milder, the infiltration was less firm, and the wandering of the disease in the lung less marked. The appearance of diarrhœa, also, seemed to have a favorable influence.

Smarkovsky,[68] of Moscow, claims that calomel given in doses of 5 or 6 cgm. (gr. $\frac{3}{4}$–1) every hour until a purgative effect is produced, is capable of jugulating croupous pneumonia and causing its abortion. This does not result, he observes, from any direct influence of the calomel upon the pulmonary lesion, but from a general antiseptic action upon the toxic material circulating in the blood, thus augmenting the resistance of the organism to the morbid local process.

Granting the major proposition, it would be more reasonable to infer that the drug acted toward limiting the production of the poison, rather than its destruction after it has reached the current of the blood.

For almost fifty years Clemens, of Frankfort, has been employing inhalations of *chloroform* in pneumonia with a view to its sedative action upon the nervous system and its anticoagulating (?) effect upon the blood. His success, as will be seen later, has been remarkable, and others following his example have obtained like results. Here again we have a powerful germicide which in this case is not only introduced into the blood, but is at the same time brought into direct contact with the culture medium itself, up to the time when complete consolidation excludes it from the air cells.

Later we have the introduction of *quinine* in large doses, given with a view to its antipyretic effect. Many authorities, including Flint,[47] believe that it is possible in this way to abort a pneumonia in its initial stage. Flint says: "As long ago as 1861, I was led by the results of the analysis of a considerable number of cases in which sulphate of quinine was given to the extent of only fifteen grains daily, to the conclusion that this remedy exerted a marked curative influence upon the disease. I can now (1881) bear testimony to the fact that, given in larger doses, namely twenty to thirty grains daily, this remedy, in a certain proportion of cases, renders the disease abortive, and that when this does not follow, the disease is often modified to a greater degree than by smaller doses. Now whatever efficacy belongs to these remedies proceeds evidently not from any direct effect upon the pulmonary affection, but from a controlling

influence over the pyrexia, thus sustaining the doctrine that the disease is an essential fever."

How this abortion is to be effected simply by acting upon the heat centre there is no attempt to explain.

Hare[*] expresses the belief that quinine with aconite or veratrum viride, employed before consolidation has taken place, has the power of so modifying the hyperæmia in the affected area as practically to abort the local process and prevent exudation.

He warns, however, against the assumption that every case if left untreated would necessarily go through all its stages. He says: "Personally I believe that it does not always run a full course and that those cases, such as Bristow mentions, which get well after the second day are instances where, from one reason or another, the infecting micoorganism is incapable of carrying out its ordinary processes.* Believing this, which is based upon the best of bacteriological investigation, we should be able so to modify the process by treatment as to partly abort the illness, provided we see the patient before the microorganism has accomplished its purpose and produced such pathological changes in the pulmonary tissues that resolution and absorption processes must take place."

A. H. Kerr[*] advocates warmly the use of *creosote* in large doses in the treatment of pneumonia. He says: "I do not say that creosote is a specific in pneumonia, but I do say, and with emphasis, that it is the nearest approach to one that has yet been reached." He dwells upon its being eliminated largely by the lungs, and thus being brought into direct contact with the lesion. It permeates the tissues rapidly, and the air at each expiration is loaded with it, as is evidenced by the odor of the room within twenty-four hours from the beginning of its use. He gives the details of a case seen within twenty-four hours after the chill. The pulse was 120, temperature 104.2° F., respiration 40. Dulness and fine crepitant râles over

* As regards this matter, we frequently hear it said of a person who has had a brief illness, that he was "threatened with pneumonia." Formerly I had little patience with this expression, believing that it necessarily meant more to the friends than it meant to the physician who employed it. But I have come to thinking that it describes very well an abortive invasion of germs, which after a brief struggle have been overcome by the resisting power of the system. For myself, I believe that scattering germs stray into the air vesicles oftener than we suppose, but not finding a suitable nidus fail to effect a permanent lodgment. How frequently we meet with a stitch in the side accompanied by some fever, and on examination are not a little relieved by the absence of physical signs. At the next visit there is scarcely a trace of the attack remaining. For one such case coming under the notice of the physician there are probably many more for which advice is not sought. A proportion of such cases are, I believe, to be accounted for in the way I have suggested.

the base of the left lung. Ten minims of creosote were given every two hours, and for the first day a grain of opium every three hours. The following day (the third of the disease) the pulse remained at 120, temperature 104°, respiration 36. Twenty-four hours later (fourth day) pulse 96, temperature 102°, respiration 26. At the next visit (fifth day) pulse 80, temperature 99°, respiration normal, patient convalescent.

Under the head of "Note di Terapia" the *Clinica Moderna*[66] states that it has been demonstrated that clysters of creosote, each containing from twenty-five to forty drops, are of great value in pneumonia, and cause a subsidence of all the alarming phenomena of the disease. (References not given.)

Robinson[67] insists strongly upon the value of creosote inhalations from the very beginning of an attack of pneumonia, not only for their beneficial effect upon the patient, but as having a tendency to lessen the danger of infection to the attendants. He provides for the vaporization of pure beech-wood creosote in considerable amount along with the production of steam. The apparatus is placed near the patient's bed, but the atmosphere of the whole room is thoroughly impregnated with the combined vapors, so that they are inhaled by the attendants as well. He is firmly convinced of the utility of this practice, which has an evident foundation in reason.

The pneumococcus, as we have already seen, is extremely vulnerable, and while it may not be actually killed by any vapor that could be safely taken into the lung, yet there is no reason why its growth should not be inhibited to a considerable extent, and the amount and virulence of the toxin materially lessened. It is no sufficent objection to this proposition that the inhaled material could not reach the interior of the air cells in the hepatized area, since all goes to show that the most active conditions for general infection are supplied by the partly filled alveoli surrounding the consolidated focus. To these cells a gaseous germicide can easily penetrate.

As to the possibility of aborting pneumonia and the value of antiseptic inhalations, Robinson says:

"One of the points in regard to the course of acute croupous pneumonia which is most important is this: to determine, if possible definitely, whether or not pneumonia has a certain well-defined course terminating in crisis on a given day, which is, so to speak, unchangeable. In other words, is pneumonia capable of jugulation? Can we abort it by any one of our known medicinal means? This question has been answered differently by different authors. Osler, for example, denies that it can. Bristow seems to be of a somewhat different opinion, since he believes that 'in very mild cases all the symptoms may subside in three days, or even two days.'

"One thing is true, however, if we may attach faith to the experimental researches of Welch, and it is that the micrococcus lanceolatus, the admitted cause of croupous pneumonia, is a pathogenic organism, one of those least tenacious of life, and that its loss of virulence is frequent and apt to occur rapidly. Is there not in this important fact a very substantial reason why we should endeavor to employ antiseptic inhalations early in the treatment of pneumonia, before lung consolidation has taken place, and while we may yet legitimately hope that the antiseptic vapor may reach every portion of the affected pulmonary area?"

Salicylates.—Robert Liegel[85] contributes a remarkable paper in which he describes a treatment which has been successful in his hands in seventy-two consecutive cases of croupous pneumonia occurring in the miners at Leoben-Seegraben. The patients ranged from sixteen years of age to seventy-four and included many subjects of anthracosis. Eight had pulmonary emphysema, six cardiac disease. A large proportion were alcoholics. The drug relied upon was sodium salicylate in "large doses," not less than 8 gm. daily. This was given in solution, with aqua menthæ piperitis to cover the taste. No other medicine was employed except perhaps an expectorant containing ipecac, if the cough was tight and racking, and small doses of morphine when the pain was excessive. Ice was applied to the head when the temperature was above 39.5° C. (103° F.).

Under this treatment not only did recovery take place in every case, but the duration of the attack was diminished one-half. In no case did crisis occur. The temperature declined from the end of the first day, until at the end of three or four days it reached the normal, and convalescence was established. The expectoration lost its distinctive character and became catarrhal, the physical signs did not fully develop, or if present, speedily retrogressed. The microscopical examination of the sputa showed a constantly diminishing number of diplococci, until, at the end of the third or fourth day, they were found to have entirely disappeared.

In the earlier cases the medicine was suspended as soon as the temperature became normal, but this was found in a number of instances to be followed by relapse; observing this, the doses thereafter were continued for some two or three days longer, and no further relapse occurred.

Previous to the adoption of this treatment the management of the disease by the usual methods had been very unsatisfactory and the mortality excessive.

Liegel considers that the salicylate exerts a specific effect, such as it exhibits in rheumatic fever. He, however, assumes that it acts

upon the mucous membrane, increasing its secretion and thereby throwing off the exudate, as croupous membrane is thrown off from

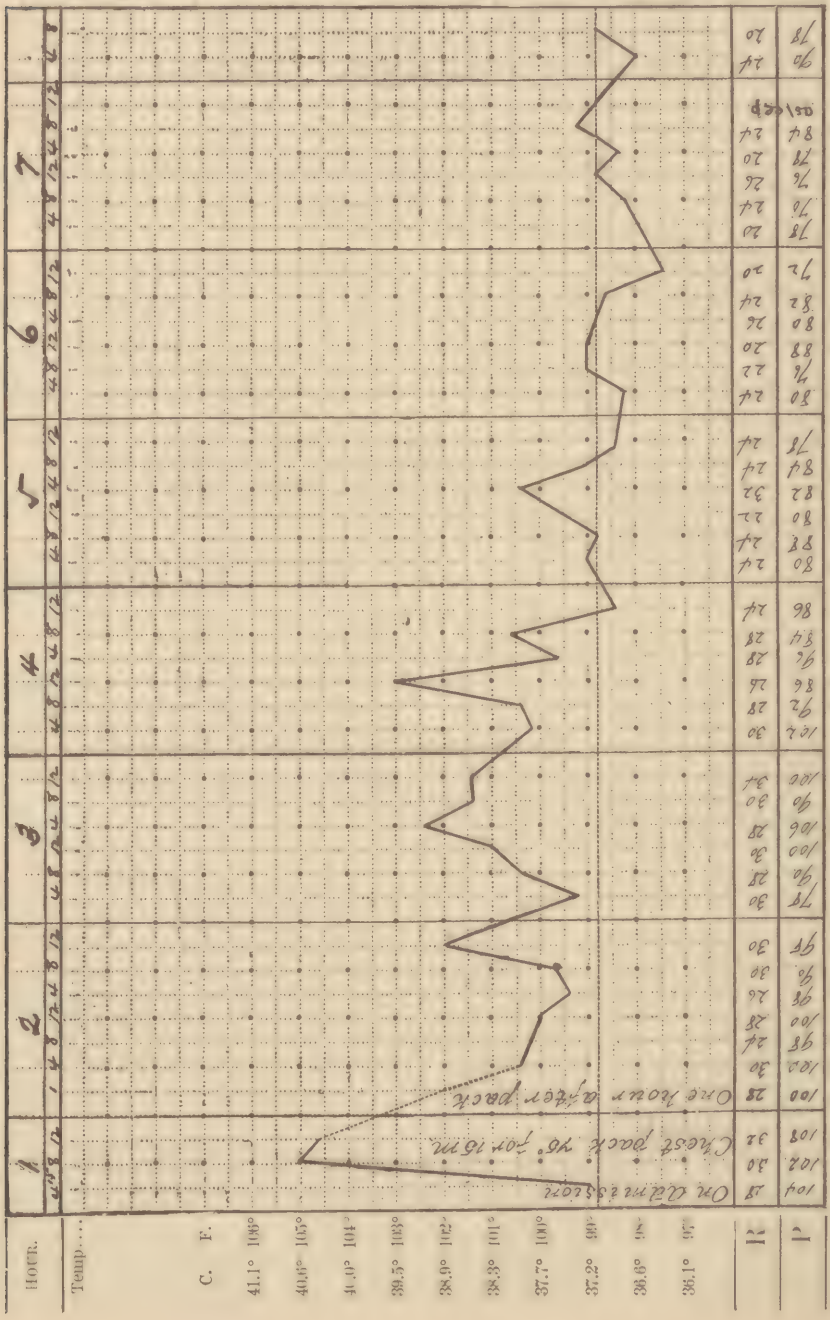

CHART No. 4.—A Case Treated with Ammonium Salicylate. Patient was admitted immediately upon the occurrence of the chill, and 20 grains of the drug were administered every four hours. Temperature reached the normal on the fourth day.

the larynx. In no instance did it give rise to any symptoms more unpleasant than a buzzing in the ears. Indeed, the dosage was fairly moderate, less than we habitually employ in the early stages of acute rheumatism, and anything like poisonous effects could scarcely be apprehended.

Liegel believes that this treatment will save nearly all cases, and he cites some instances to show that the most unfavorable conditions may be recovered from under its use. In one of these a man sixty-seven years of age was taken with pneumonia while living in a damp cellar, in which six persons were huddled together. He had been insufficiently nourished before the attack, and when first visited was found delirious and too much prostrated to be removed to the hospital. He recovered, however, in the same time as the others, notwithstanding his miserable surroundings.

This is the latest and most important illustration of the possibility of so acting upon the blood as to hinder the growth of the bacteria, and to diminish, if not prevent, the formation of toxin.

The following is from a private letter to the writer, from Dr. A. Ross Matheson, of Brooklyn. The substitution of the ammonium salicylate for the sodium salt has much to recommend it, but the latter has seemed to me to be better borne by the stomach:

"For several years I have used the salicylate of ammonium in pneumonia, more especially in those cases occurring during the grippe season and in which there was a grippe element more or less pronounced. I am now treating a case of lobar pneumonia in the third stage in which the only remedies administered have been salicylate of ammonia, codeia sulphate, and strychnia sulphate. The symptoms in this case in the beginning were formidable in the extreme, but modified early as a result, I believe, of the effects of the ammonium salicylate. I administer it in ten to fifteen grain doses, and endeavor to have the patient take from one and a half to two drachms in twenty-four hours.

"I find that ammonium salicylate has some advantages over the soda salt. It is stimulating, while the latter is more or less depressing, and it does not produce to the same extent the throat and ear disturbances. I am satisfied that it has decided value in the treatment of pneumonia."

Dr. J. H. Ferguson, of Mine La Motte, Mo., informs the writer that he has records of one hundred and six cases of pneumonia treated by him with sodium salicylate, showing one hundred recoveries and six deaths. He was led to this method by the belief that inflammation in general was intensified by uricacidæmia. He gives five grains every two hours, together with potassium citrate, to stimulate the kidneys.

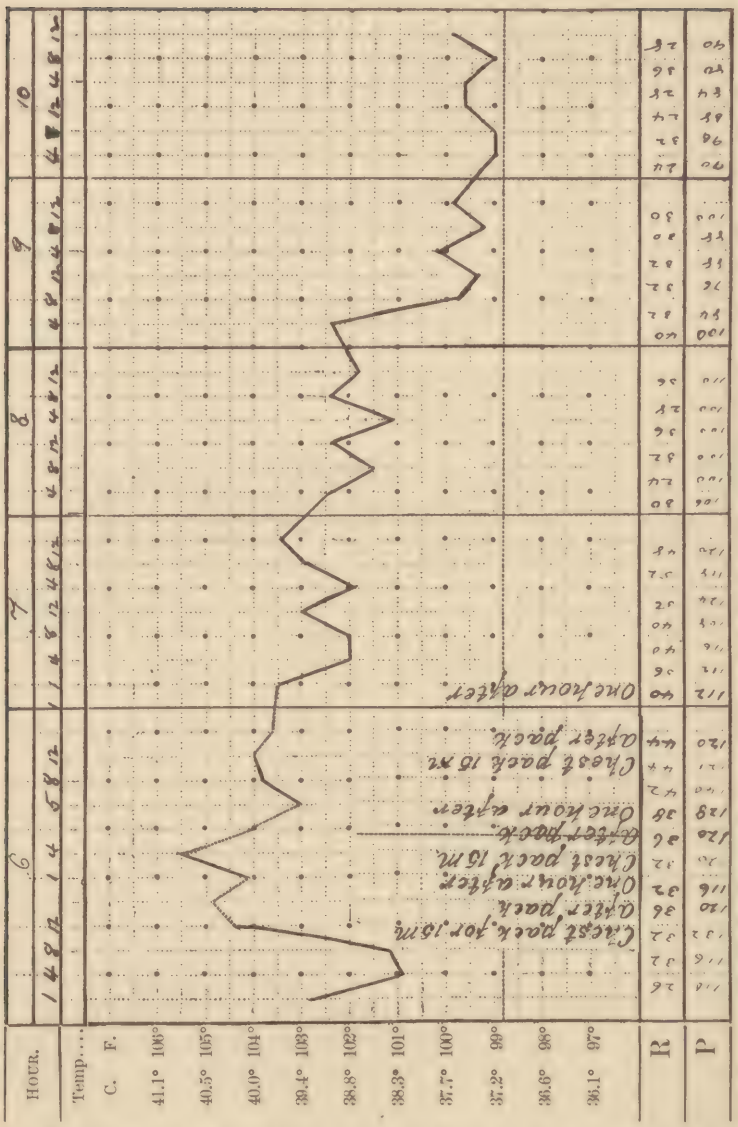

Chart No. 5.—Patient having Double Pneumonia who was Treated with Sodium Salicylate. Patient entered hospital on the third day of the disease with pneumonia of the lower right lobe. On the sixth day the left lower lobe become involved. Cold packs applied to chest for temperature above 103.5°, and 20 grains of sodium salicylate administered every four hours. Defervescence by lysis. Normal temperature on tenth day.

Salicylic acid has been employed as an antipyretic in pneumonia by different clinicians, but has not been found to possess special advantages over other remedies of that class. It has not, I believe, been used in this disease with a view to its antimicrobic effect.*

* According to Buchholz 0.15 per cent. of salicylic acid will prevent the development of bacteria in ordinary organic mixtures, and the influence of 0.005 per cent. (1 : 20,000) is plainly visible. Sodium salicylate has about the same power.—Wood's "Therapeutics, its Principles and Practice," 10th ed., Philadelphia, 1898, p. 705.

From the foregoing facts and opinions, as well as from the theoretical considerations already brought forward, it seems clear that there is ample justification for a treatment of pneumonia based largely upon the causal indication. In other words, we may reasonably expect benefit in a considerable proportion of cases from the use of means addressed directly to the germ present in the lungs. The practical question to be solved is what agent will act most powerfully upon the specific organism with least inconvenience or danger to the patient. This opens a wide field for study and observation upon which I firmly believe it is the duty of the profession to enter.

Thus far the most efficient and least harmful drug appears to be the salicylate of sodium, as employed by Liegel. As we have seen, seventy-two consecutive cases, many of them most unpromising, have been treated with it without a death. This is a very remarkable record, and certainly recommends the treatment most strongly for further trial. Apart from the experience of Liegel, it does not appear unlikely that a drug which is capable of producing such decided results in acute rheumatism should be effective against an organism so sensitive as the pneumococcus.

Liegel did not observe any depressing effect from the salicylate in the doses he employed (8 gm. daily), but such if apprehended could be guarded against by the use of strychnine, etc.

Creosote has been found useful by several observers and deserves further trial. It may be given by the mouth or the rectum, or employed by inhalation. The dose by the mouth is ten to fifteen minims every two or three hours, largely diluted; by the rectum twenty-five to forty minims in a clyster. By inhalation it is useful only as an adjunct.

The following case, in which creosote was employed, was seen by the writer in consultation with Dr. Bainbridge, of this city, January 9th, 1899.

Miss C——, aged 46, had suffered from influenza in November, 1898, and from a second attack beginning January 1st, 1899. She was first seen by Dr. B. January 6th, when signs of pneumonia were present in the right base. On the seventh, there was complete consolidation at the right base with beginning signs in front over the right lower and middle lobes. Pain in bones, relieved by a few doses of phenacetin and salophen.

On the 8th, nitroglycerin and sparteine sulphate were begun. Alcohol, which seemed to be indicated, caused marked cerebral excitement. The right middle and lower lobes were solid.

On the 9th, the temperature reached 105° F. and the writer was called in consultation. The treatment agreed upon included strychnine sulphate by the mouth, and creosote, 20 minims, with sodium bromide, 25 gr., by the rectum. This was repeated every four hours,

the creosote and bromide being mixed with the white of an egg to which three teaspoonfuls of brandy were added.

On the 10th, a small area of beginning consolidation appeared at the angle of the left scapula, and a few râles were heard in the left axilla. The enemata causing discomfort, the bromide was reduced to 20 gr. and the creosote to 10 minims.

On the 11th, there were signs of pleurisy in the left axilla. Later, a brief attack of pulmonary œdema occurred, and the pulse rose to

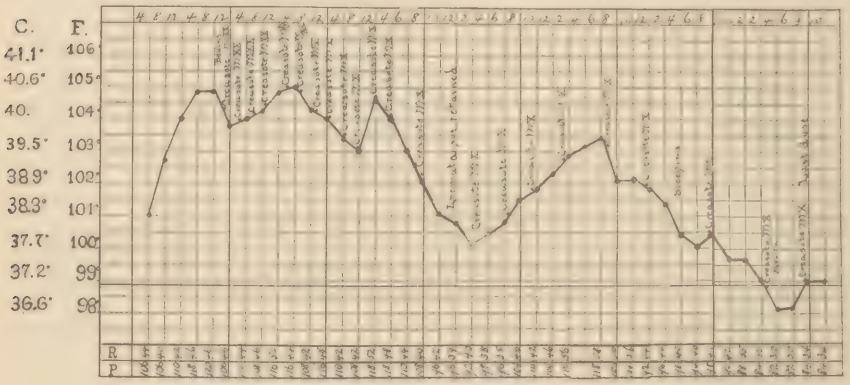

Chart No. 6.

130 and the respiration to 60. The attack yielded to brisk counter-irritation and inhalations of oxygen. During the night of 12th–13th, the patient was greatly depressed and the heart was very weak. Stimulants increased.

On the 14th, the patient was very comfortable; convalescent.

In this case there was pseudo-crisis on the sixth day, with subsequent rise of temperature and defervescence by lysis, the temperature reaching the normal on the eighth day (see chart No. 6).

Carbolic acid may perhaps be found to have some advantages over creosote. It is less disagreeable to take, and probably less irritating to the stomach. It is readily taken into the system, as is shown by its speedy appearance in the urine. The usual dosage may be greatly exceeded with perfect safety. Wigglesworth[29] gives as much as six grains every two hours day and night in the treatment of scarlatina, continuing it until the risk of infection disappears. Dr. Jackson, of Alnwick, Eng., in a letter to the writer, says that he gave to a patient suffering from charbon three drachms in the first twenty-four hours, two drachms in the second, and one and one-half drachms during each day thereafter till the end of the fortnight. It was retained when everything else was vomited, and no ill effect was experienced except a moderate albuminuria* which disappeared

*This may have been the result of the infection, and not of the acid.

in a few weeks. The patient was rescued from an apparently hopeless condition.

I have myself given one hundred and thirty to one hundred and fifty minims of the liquefied acid in twenty-four hours, for several days, without the least unpleasant result. The urine had a slightly greenish color when voided, and became perfectly black after standing a few days, but contained no albumin. Declat cured intermittent fever with carbolic acid when quinine had failed, and if it is capable of destroying the plasmodium in the blood it would doubtless be capable of inhibiting the growth of the pneumonia germs in the alveolar contents.

Corrosive sublimate is believed by many to act favorably in diphtheria when taken in quantity sufficient to act upon the whole mass of the blood, but yet within the limits of safety. We have already seen that there is evidence that calomel in a large dose may be very useful in pneumonia, and it is quite possible that still better effects might be obtained from the more soluble salt. Skoda employed it, sixty years ago, in pneumonia, with a mortality of 14.5 per cent.[2]

Almost immediately upon the discovery of the anæsthetic property of *chloroform*, Clemens,[70] of Frankfort-on-the-Main, employed it to relieve the pain and shortness of breath belonging to the early stages of pneumonia. His success with it was great, and he was still employing it at the date of his last communication in 1889. He claims that he has given it in every case of pneumonia occurring in his private practice since about 1850, and with such success that he has not lost a single patient. He attributes this to the soothing effect upon the nervous system, the lessening of shock, and the freer respiration resulting from the absence of pain. But he states that in addition to these effects there is often a marked hastening of the crisis, and that the average duration of the disease is lessened. His explanation is that the chloroform defibrinates the blood in the lungs and prevents hepatization.

Baumgartner was one of the first to make extensive trials with inhalations of ether and chloroform in pneumonia, and found that in this as in other chest diseases the feeling of constriction, the stitch, the expectoration, and the sleeplessness were relieved. At the same time the abundant secretion from the bronchial membrane which, especially in the case of ether inhalation, marked the beginning of the treatment subsided as the treatment progressed. Wucherer published a pamphlet on the subject as early as 1848, and Varrentrapp and Thiele treated a great number of cases of pneumonia with inhalations of chloroform.

Oertel[71] found decided benefit from inhalations of chloroform,

and refers to the experience of Baumgartner, Wucherer, and Varrentrapp, Thiele, and others which agree as to the relief which may be afforded by their use. Oertel employed them largely in Pfeiffer's clinic in Munich, from 1860-1863, mostly in the advanced stages of the disease, about the fifth or sixth day, in cases in which there were extensive hepatization and marked involvement of the pleura, rendering the breathing irregular, frequent, and superficial; when the expectoration was more or less suppressed, and the viscid exudate obstructed the air tubes; when coarse râles were heard over large areas, and when rapidly increasing cyanosis indicated insufficient aeration of the blood. Frequently the inhalations were repeated as often as five or six times in twenty-four hours and pushed to commencing narcosis. The results were most satisfactory. The respiration became deeper; the pleuritic pain was relieved, the cough was lessened, and the sense of oppression in the chest disappeared. These results were maintained for a longer or shorter period after each inhalation, and were not entirely lost during the intervals. The râles were diminished; a more or less free expectoration was established; cyanosis was less marked; and for the most part the treatment was successful in bridging over the critical period. Oertel sums up his experience as follows: "I consider the inhalation of chloroform when the above indications are present as a means of treatment in pneumonia that would be difficult to replace by any other."

As a contraindication he regards disposition to headache, vertigo with confusion, decided congestion of both lungs, and finally general depression. He insists that if the inhalations of chloroform are to be of real value in the treatment of pneumonia, they must be administered by the physician himself.

Now in view of the hastening of the crisis and the shortening of the attack observed by Clemens, and the general improvement of the patient's condition so strongly insisted on by Oertel, it may fairly be asked whether these inhalations of chloroform do not have a direct effect upon the exudate to render it an unfit medium for the culture of the pneumococcus. Experiments conducted by Dr. J. S. Thacher, Pathologist at the Presbyterian Hospital, New York, at the suggestion of the writer, showed that this organism was very sensitive to chloroform. The penetration of chloroform vapor to all parts of the lung is attested by the familiar facts of anæsthesia. Nor is it impossible that the whole mass of the blood should become so impregnated with the agent as to render the exudate from it an unfriendly medium for the growth of the coccus. We know how in the case of anæsthesia by ether the whole body, solids as well as fluids, becomes permeated by the odor, and how long a time elapses before it ceases to

be apparent in the breath. Everything leads us to suppose that the diffusion of chloroform through the system is equally complete, though its odor being less powerful, we have not the same evidence of its presence.

For the purpose in view it would by no means be necessary that the life of the organism should be destroyed, but only that its multiplication should be prevented, something which is very much more easily accomplished.

I have found that chloroform may advantageously be given in connection with oxygen by adding one or two drachms to the water in the wash-bottle. Though the chloroform settles at once to the bottom of the bottle, the agitation caused by the bubbling of the gas through the water insures the vaporization of enough to produce a considerable narcotic effect. Aside from any action upon the germs, the chloroform allays pain, soothes nervous agitation, and promotes sleep. This method of administration will be sufficient when only a moderate effect is desired, but if the abortive action of chloroform is aimed at, a more concentrated vapor will need to be inhaled.

Other substances besides chloroform have been proposed as inhalants. *Carbolic acid* from its volatility as well as its germicidal property would suggest itself at once. So far from being an irritant, it is, when properly diluted, a local anodyne, and its vapor can be taken into the lungs in considerable strength without causing inconvenience. In some experiments I have made carbolic acid was used with chloroform, and apparently with good effect.

The effect of *quinine* in large doses has been already considered. It has been given for its antipyretic effect and as a tonic to increase the vital resistance. Much more powerful antipyretics and equally good tonics, however, have failed to exhibit the same beneficial action in pneumonia. But we know how quinine enters into the blood and destroys the malarial organism, and with this demonstration of its germicidal power we can well understand that other germs as well as the plasmodium may succumb to it. If given with a view to abort the attack the daily dose should be not less than 30 to 40 gr. If the resulting cinchonism is severe, it can be relieved by full doses of one of the bromides, preferably, in this case, the bromide of ammonium.

The attempt to cut short an attack of pneumonia will be more likely to be successful the earlier it is begun. If it is determined to adopt this line of treatment it would be well to begin with the administration of 20 to 30 gr. of calomel.* This will at least insure a thor-

* The calomel should be placed dry on the tongue and washed down with as little

ough cleansing of the alimentary canal, and probably will also increase the secretion from the liver and facilitate the portal circulation. These will be points of vantage gained, and if, as will be likely to be the case, it is followed by a considerable fall of temperature, it will open the way to the use of other germicides. The choice among these will be decided by the preference of the individual practitioner. It need not necessarily be limited to a single agent. Thus sodium salicylate, which has more or less of a depressing tendency, may be given by the mouth, while at the same time the patient is receiving enemata of creosote, which in medicinal quantities is a stimulant. Or, while a germicide is taken by mouth or rectum, chloroform may be given by inhalation. This will be specially indicated in cases in which the pain is very severe or insomnia is present.

While treatment of this kind is most likely to be effective if early resorted to, yet it should be attempted at any time during the pyrexia. For we can never appreciate how much of microbic activity may be going on outside of the hepatized area, either without physical signs or with only the signs indicating congestion.

It is not to be expected that an obvious result will be obtained within a few hours. In Liegel's cases the temperature remained unchanged until the end of the first day, and when abortion has followed the use of other methods the same delay has usually been observed. Even if it were possible to immediately cut short the production of toxin, some time would be required for the elimination of that already in the blood, and meantime the fever would continue.

Next to attempts to inhibit directly the growth of the bacterium and the formation of toxin, the local indication is to obviate as far as possible the effects of the obstruction of respiration and circulation. These two forms of obstruction are so closely allied in their results that for our present purpose they may properly be considered as one. They play a very important part in the history of pneumonia, causing directly or indirectly a greater share of the mortality than is caused by the toxins circulating in the blood.

Bloodletting.—In no disease was the lancet employed during the first half of the present century more freely and more indiscriminately than in pneumonia. The theory then in vogue as to the nature of the disease made this treatment seem natural, and it must be admitted that in the majority of cases many of the symptoms were alleviated for a time after a copious bleeding. When this was resorted to early, the pain was relieved, the breathing became freer and less frequent, and

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water as possible. By withholding fluids for about three hours the danger of excessive purging will be avoided.

the pulse lost the hard, tense character supposed to be so typical of inflammation. The temperature in those days was not noted. Then, too, the thickness of the "buffy coat" and the "cupping" of the clot were pointed to as showing how urgent had been the necessity for letting blood. Balfour[73] gives a very vivid picture of this treatment in his presidential address before the British Medical Association, session of 1898.

It cannot be said, however, that the testimony even of the writers of that day was very favorable to the practice. Thus it is recorded that in Berne, in 1762, ninety-five cases of pneumonia were treated by bleeding with eighty-five deaths, while of seventy-seven treated without bleeding only ten were fatal. But these bleedings were practised for the most part early in the attack, and before the pulmonary obstruction became a prominent factor in the case. As a means of relieving this latter condition the practice is once more gaining in favor.

Osler[23] says: "To bleed at the very onset in robust, healthy individuals in whom the disease sets in with great intensity and high fever is, I believe, a good practice. I have seen instances in which it was very beneficial in relieving the pain and the dyspnœa, reducing the temperature and allaying the cerebral symptoms."

And although the lancet has so far passed out of use that many a practitioner of the present day would scarcely know how to open a vein, it is by no means certain that a good many lives might not be saved by such a timely and judicious bloodletting as would relieve the overdistended venous system and give a measure of ease to the laboring right ventricle. Many recent writers favor the practice under certain conditions. Osler took from 25 to 30 oz. of blood in cases which he thought it indicated, but adds that he had seen only one of twelve or fifteen cases so treated that recovered. Maragnioni Felice and Cantani resort to it sometimes for its hydraulic effects. Bianchi favors it, especially in children, when great dilatation of the right auricle is present. Liebermeister upholds it when œdema occurs in the unaffected lung. Fowler[73] says:

"Cyanosis and signs of overdistention of the right side of the heart with epigastric pulsation and prominence of the jugular veins and a small and irregular pulse, are indications for venesection, and relief is generally given when 6 to 8 oz. of blood have been withdrawn. The improvement is perhaps most obvious in cases accompanied by or following bronchitis, but unfortunately it is as a rule of only short duration."

Were it not that much the same result may be brought about with less drain upon the system, as we shall see hereafter, the indication

would, no doubt, often be present for the abstraction of blood, and as it is, whenever it becomes evident that the right heart is unduly distended and is beginning to flag in its efforts to force so large a mass of blood through the obstructed pulmonary capillaries, and when adequate relief is not obtained by diverting a greater proportion of the blood into the arteries, it is the duty of the physician to open a vein and allow blood to flow until the venous tension is relieved. In children, however, the indication may be better met by the use of a number of leeches proportioned to the age and strength of the patient. Still, bloodletting is, at the best, a measure to be adopted only in extreme cases, and, like any remedy so employed, cannot be expected to produce brilliant numerical results. But, if it saves only a few otherwise hopeless cases its use is amply justified.

Nitrites.—Except in some extreme cases, however, most of the good and none of the bad effects of bleeding may be obtained by withdrawing blood from the veins and storing it up in the arteries. This is effected by the use of that class of medicines which have the power of relaxing the arterial system. Chief among these are the nitrites, but the property is shared in a less degree by numerous other remedies, notably by aconite, the value of which in inflammatory diseases accompanied by high arterial tension is now universally admitted. It was in connection with this drug that the idea of "bleeding a patient into his own vessels" was first suggested, and the phrase well expresses the peculiar action of this class of medicines. By their specific effect upon the vasomotor nerves they cause a relaxation of the musclar coat and a consequent dilatation of the whole arterial system. The change in capacity which may be effected in this manner is much greater than is generally believed. On this point Ringer says: "It has been shown that the vascular system is always in a state of semicontraction, and that by paralyzing the vasomotor nerves it is possible to double its capacity." An effect much short of this would be sufficient to produce a vast difference in the dynamics of the circulation, and to afford a large measure of relief to the overdistended venous system.

It is in this way, I contend, that we should direct our efforts in cases involving pulmonary obstruction. I am aware that there is an opinion prevalent that these drugs are contraindicated when there is feebleness of the heart's action; and there are many practitioners who would regard with a feeling akin to horror the administration of nitroglycerin, for example, when the pulse is notably small and frequent. If the condition broadly but vaguely described as heart failure were thought to be impending, the administration of such a drug would be looked upon by them as a sort of *coup de grace*. And

so it might prove to be if the feebleness of the pulse were due simply to general adynamia. But the case is altogether different when it is a mechanical rather than a vital condition we have to deal with. By increasing the capacity of the arteries we relieve the veins by exactly the amount of this increase. Lessening the pressure in the veins lessens *pari passu* the pressure in the pulmonary circulation, and with it the tumefaction of the mucous membrane and the tendency to exudation into the air passages. Relief to the respiration and lightening of the labor of the right heart are the immediate consequence.

The most prompt in its action among the nitrites is that of amyl, but its influence is so brief that it is unsuited except for extreme emergencies. Nitroglycerin acts also with great promptness, especially when given under the skin, and its effect is maintained for forty-five to sixty minutes. In doses of gr. $\frac{1}{50}$ up to $\frac{1}{10}$ it may be given without hesitation, and repeated every half-hour if required. Its effect when cyanosis is present, and the chest is filled with râles, is often most satisfactory. The lividity clears up, the râles diminish, the respiration becomes deeper and less frequent, and the pulse larger and slower. At the same time the sensorium recovers from its oppression. These results may be obtained to some degree in a period of not more than ten minutes. The dose may require to be repeated several times at intervals of fifteen to thirty minutes.

With the remedy thus administered all the advantages of bloodletting may be secured and without the loss of vitality that bleeding necessarily entails. When the conditions calling for this form of relief are more permanent it is well to employ a more lasting influence, such as is afforded by sodium nitrite. This salt is slower, but at the same time more persistent, in its action; and by doses of one or two grains every two hours the vessels may be maintained in a relaxed condition which can be added to at any time by supplementary doses of nitroglycerin should occasion require. I think it is difficult to exaggerate the value of this method when the right heart is in danger of breaking down under the strain imposed upon it. I have repeatedly seen patients rescued by it when death seemed immediately impending.

As to the dosage of nitroglycerin, the drug may be given much more freely than it usually is, provided the patient has been found, by testing with small doses, not to possess an undue susceptibility to its effect. On this point Armstrong[74] has contributed a valuable article. He cites a case mentioned by H. C. Wood, in which gr. $\frac{1}{100}$ produced insensibility, and another case in which gr. $\frac{3}{50}$ caused complete unconsciouness and loss of the radial pulse. Such cases are

due to idiosyncrasy of the patient, and not to the legitimate action of the drug. On the other hand, he mentions instances in which enormous quantities, even to 76 grains in twenty-four hours, were taken with only beneficial results. Nitroglycerin is a remedy of which tolerance is rapidly established; and Armstrong is no doubt right in his suggestion that tolerance is increased by a previous condition of vascular tension.

Practically, in pneumonia, the dose must be graduated in each case by the results obtained. So long as there is evidence on the one hand of venous repletion, and on the other of relief of this condition by the use of the drug, we are safe in pushing it without much regard to the amount given. If under its use the color of the face and lips improves and the respirations become less frequent, the indication is to continue it in the doses necessary to maintain this effect. These will seldom exceed gr. $\frac{1}{2}\frac{1}{5}$ every hour, though in extreme cases this quantity or even more may be repeated every half-hour, or even oftener.

Oxygen.—Inhalations of oxygen are now very commonly resorted to in chest affections when the dyspnœa becomes severe. The use of this agent was introduced into this country by the writer in 1860. By experiments on animals he had demonstrated that by enriching the atmosphere with additional oxygen, life could be maintained for long periods under conditions of tracheal obstruction that would be immediately fatal in common air.

The introduction of compressed oxygen in cylinders for commercial purposes facilitated the use of the gas in sufficiently liberal quantities to test its real value as a therapeutic agent, and for the last twenty-five years it has occupied a prominent place in the treatment of respiratory affections and especially of pneumonia.

Its value in this disease rests on a broader foundation than is immediately apparent. It is not alone that it tends to avert suffocation. Indeed, it is very seldom that a patient with pneumonia dies directly from deprivation of oxygen. As a rule, before death from actual suffocation takes place, the heart, and particularly the right heart, gives out; and the fatal result is from asystole. This exhaustion of the right heart is a gradual process, and is brought about by the increased muscular effort required to propel the blood through the affected lung. The pulmonary ischæmia is made up of two factors, one of which is the obstruction of the vessels, and the other and very important one is the sluggishness with which blood not duly aerated circulates even through unobstructed capillaries.

It is in the relief of this latter condition that oxygen is most valuable. The moment the arterialization of the blood is improved the

circulation becomes easier and the labor of the right heart is lightened. It is a serious error, however, to defer the use of oxygen until the dyspnœa has become urgent. By its timely employment the cardiac force can be conserved and congestion of the unaffected lung territory is in a great measure prevented. But inasmuch as blood, even under the most favorable conditions, will not take up an excess of oxygen, if we delay too long and suffer the access of air to become too much restricted, no addition of oxygen acting upon the limited quantity of blood circulating in the unaffected area will suffice to restore the balance and regain the ground which has been lost.

While the results obtained from oxygen in the croupous form of pneumonia may not be so favorable as in the bronchial, still it is capable of rendering valuable aid in a large proportion of cases. A common source of disappointment lies in reserving its use until a period when, for reasons already considered, its value is comparatively limited. Even in these cases it may serve to bridge over a time of special danger, but the best results are obtained when it is given more or less freely from the moment that it becomes clear that the case is one of more than moderate severity. Its good effects will be shown in lessened frequency of the pulse and respiration, a better color of the face and lips, and fewer moist râles in the chest.

In using the compressed gas it is allowed to escape from the cylinder through a wash-bottle, the valve being so adjusted that the gas bubbles gently through the water. From the wash-bottle the gas is carried to the patient's mouth through a flexible tube provided with a mouth-piece of glass or hard rubber. If the patient is in a condition to hold this in his mouth, no more will be required. Otherwise it must be held by an attendant in such a position that the escaping gas will be drawn into the lungs with the current of inspired air.

If the patient is comatose a small flexible catheter may be passed through one nostril into the nasopharynx, and connected with the wash-bottle. In this way very little of the gas is wasted.

If it is desired to add any volatile substance to the gas inhaled, a solution containing it may be made to replace the water in the wash-bottle. Chloroform may be employed in this way.

There is no advantage in a lavish use of the gas, as the blood will take up only a very limited amount. If it escapes too freely, it adds to the sensation of breathlessness, as is the case when one faces a strong wind.

Only pure oxygen should be employed. The addition of nitrous oxide with the idea that it is more soluble in the blood should be condemned, as the oxide is useless for the purpose of respiration and interferes with the proper interchange of gases in the lungs.

In pneumonia the indication for a resort to oxygen is present as soon as the respirations exceed thirty-five per minute, and earlier than this if mucous râles develop outside of the area of consolidation, or if the lips assume a dusky hue. Under these conditions it may be given continuously, or during a prescribed number of minutes in each hour.

Treatment of the General Condition.

To combat the toxæmia we have as yet no direct means, unless it be in serum therapy, the value of which is still *sub judice*. We may hope to lessen the production of toxin by means which we have already considered, but for relief from the effects of what, in spite of our efforts, finds its way into the circulation, we must still rely upon the emunctories. The chief of these are the skin, the kidneys, and the intestinal glands. The activity of these is best promoted by the liberal use of simple cold water. This should be given as freely as the patient will take it, or if he be delirious or unconscious it should be administered at intervals in the same manner as his food.

Cathartics.—Additional stimulation of the intestinal glands may be advisable, employing cathartics selected especially with that view, and particularly calomel if it has not already been given for its abortive action. It is desirable that the bowels should be kept as free as may be without too great a drain upon the general strength, not only as promoting elimination, but as guarding against abdominal fulness and the consequent hindrance to respiration.

Diuretics.—The kidneys often fail to excrete the normal amount, indeed the urine is generally scanty, and though of high specific gravity contains much less than the normal amount of solids. Thus the most important channel of elimination is choked and its efficiency greatly impaired precisely at the time when a depurative action is most needed.

The importance of stimulating the kidneys during the pyrexia of pneumonia has not been recognized as fully as it should be. Whether with this object in view or not is not stated, but Nilsson[75] treated fifty-eight cases of pneumonia with potassium iodide, and with only three deaths. His death rate with other methods had ranged in different years from twice to three times as great. He gave 1 gm. (15 gr.) every three hours day and night; on an average each patient consumed from 40 to 50 gm.

Potassium iodide in these doses is a most efficient diuretic and it does not irritate the kidneys, as is shown by the enormous doses given with impunity in syphilitic disease. It is therefore well adapted

for use in pneumonia when a diuretic is indicated. It can very well be given by the rectum, thus saving the stomach for other uses.

Spirit of nitrous ether is also an available diuretic, and acts at the same time as a diaphoretic, a vasodilator, and a general stimulant. This combination of properties fits it admirably for use in the stage of pyrexia when the skin is dry, the urine scanty, and the pulse small and weak. It may be given in teaspoonful doses largely diluted with water, and repeated at short intervals.

Diaphoretics.—If the skin remains hot and dry notwithstanding these measures, the wet sheet applied with a blanket over it in the manner recommended by Baruch should be resorted to. This seldom fails to excite perspiration, accompanied by some degree of reduction of temperature and a marked relief of the nervous and cerebral symptoms. But the cold bath as applied in typhoid fever, with the subsequent exposure of the patient to the temperature of the room with only a sheet to cover his otherwise naked body, is a measure not to be recommended. A recession of the blood from the surface to the already congested interior can scarcely fail to be the result, and I have found the feet and legs cold under these conditions when the rectal temperature was in the neighborhood of 105° F.

The diaphoretic action of the wet sheet may be aided by the administration of Dover's powder. Pilocarpine has been recommended, but the danger that it may induce pulmonary œdema has prevented its being tested to any considerable extent.

Aside from special modes of treatment, which will be considered hereafter, the general principles to be kept in view are to sustain the heart, facilitate respiration, and husband the nerve force.

Cardiac Stimulants.—The tendency to death in pneumonia is in the direction of cardiac failure. This failure is due partly to direct toxic action upon the cardiac nerves and the heart muscle. Exhaustion as a cause of heart failure is limited principally to the right ventricle. The increased effort required to propel the blood through the obstructed pulmonary vessels continued through several days necessarily fatigues the muscular fibre, and this fatigue often goes on to the extent of paralyzing the ventricle and causing asystole.

The action of the pneumotoxin in inducing heart failure extends to the left heart as well as the right, but its effect upon the former is less disastrous as the labor required of the muscle is less severe; in fact, it may be abnormally light, owing to a diminished supply of blood received from the lungs. This is the case whenever pulmonary obstruction is a marked feature and dyspnœa a prominent symptom. When the toxic influence is pronounced, there may be a paresis of the vasomotor system, as claimed by Romberg.[76] His ex-

periments on animals lead him to believe that the circulatory embarrassment in such cases is due not so much to primary impairment of the heart's power as to vasomotor relaxation with secondary cardiac failure when the blood pressure falls below a certain point.

Van Santvoord,[77] in a series of cases of pneumonia observed in the winter of 1897-98, was able to verify the fact of this low pressure in strongly toxic cases, by means of sphygmographic tracings. He therefore claims that the first indication, viz., to sustain the action of the heart, is best met by the use of such medicines as increase arterial tension. Foremost among these he would place digitalis.

But granting the low tension, the writer believes, for reasons already given, that the condition is a conservative one, and that the welfare of the patient, so far from being promoted by a general narrowing of the arteries, would be distinctly jeopardized thereby. If the object in view were solely or principally the increase of arterial tension ergot would be the better agent.*

As a heart stimulant in these cases strychnine takes the foremost rank. Its use in small doses may be begun quite early with advantage. At first not more than gr. $\frac{1}{60}$ may be required, to be repeated every four hours. Later, $\frac{1}{50}$, $\frac{1}{40}$, $\frac{1}{30}$, and even $\frac{1}{20}$ may be advantageously employed. If necessary the remedy may be pushed to the point of producing muscular twitching. Short of this point the degree to which the nervous system is under the influence of the drug may be roughly estimated by noting to what extent the knee-jerk is increased.

When extreme cardiac stimulation becomes necessary and strychnine cannot be further pushed, Horatio Wood recommends to alternate it with cocaine.

A good article of tincture of strophanthus will give all the desirable effects of digitalis without its action upon the vasomotors. Its power is expended upon the heart muscle and not upon the vessels.

* As to any aid to the heart to come from increase of arterial tension it is difficult to see by what mechanism this aid is to be attained so long as the venous blood is dammed up in the lungs. Doubtless increasing the power of the circular fibres, or what is the same thing, diminishing the calibre of the arteries, will enable the left ventricle to transmit the force of its contractions more directly to the capillaries and through them to the venous system. But what is the advantage of this when it serves only to distend the veins more completely and adds nothing to the power that moves the venous current? For we know that *all* the blood in the body can be accommodated in the venous system alone, and is so accommodated from the moment the heart ceases to beat. Since, then, the simple retractility of the elastic coat, without any muscular aid, is sufficient to empty completely the whole arterial system, why force the muscular coat to aid in doing what is already only too effectively accomplished?

It is much quicker in its action than digitalis, its effect upon the pulse rate being noticeable within an hour. Fraser[7] considers its action upon the heart to be many times greater than that of digitalis. It possesses a very decided diuretic power, which is of no small value when the urine is scanty and the kidneys are not active in carrying off the toxin accumulated in the blood.

Tincture of strophanthus may be given in doses of four to eight drops, hypodermatically if necessary. Indeed, in all urgent cases, the cardiac stimulants capable of being administered in this way should be so employed.

Delafield, in his lectures at the College of Physicians and Surgeons, New York, asserts his belief that convallaria exerts a relatively greater action upon the *right* side of the heart, and he therefore recommends its use when the pulmonary circulation is obstructed.

Other cardiac remedies are caffeine, sparteine, adonidine, theobromine, etc. Caffeine is capable of rendering good service, the others are seldom employed. A clyster of half a pint of hot strong coffee, strongly recommended by Beverley Robinson, I have often found extremely useful. From the first the use of coffee as a beverage is to be encouraged unless contraindicated by sleeplessness.

Belladonna is of special value at the crisis to combat the tendency to collapse with cold perspiration that seems to be brought about by withdrawal of the stimulus of fever. It is indicated whenever the hands are cold and moist, the pulse is small, the senses are dulled, the brain is torpid. It is best administred as atropine, and hypodermatically.

Alcohol.—In all cases with an asthenic tendency alcohol should be resorted to in good season. The dose at first may be small, perhaps only two or three drachms of brandy or whiskey or an equivalent amount of wine, every two or three hours. But if the pulse grows smaller, and its frequency rises out of proportion to the temperature, the quantity should be increased so long as by so doing these symptoms are held in check. Particularly in subjects habituated to the use of alcohol a liberal use of this stimulant will be required. There can be no greater mistake than to put a patient suffering from pneumonia upon half an ounce of whiskey every two or three hours when he is accustomed in health to consume the better part of a pint every day of his life. The very frequent supervention of delirium tremens in the course of pneumonia is probably due to the shock of infection acting upon the brain cells already enfeebled by the chronic effect of alcohol. It is not safe to attempt its management under these circumstances by withdrawing the stimulant, as is usually done in uncomplicated cases. Enough alcohol must be given to avert the collapse

which will ensue if to the depressing effect of the toxæmia a marked reduction of the accustomed stimulation is added. Each case must be studied by itself. Tremor, wakefulness that cannot be overcome by a reasonable use of hypnotics, a dry tongue, a rapid, feeble pulse, coarse râles in areas not invaded by the pneumonia, all these call for a liberal yet cautious use of alcohol, supplemented, of course, by the drugs already mentioned.

The same is true of marked delirium apart from alcoholism. This symptom nearly always calls for alcohol when it occurs after the first two or three days. Delirium at the outset of an attack of pneumonia may depend upon individual peculiarity and have little significance. The routine practice of applying an ice-bag to the head in every case in which there is active delirium is scarcely to be commended. Not seldom the cerebral excitement is the result of anæmia or ischæmia of the brain, and it will then be made worse instead of better by the application of cold.

Very large quantities of alcohol are sometimes given with good effect in apparently desperate cases. It is remarkable that no intoxicating effect is produced by an amount that would overwhelm a person in health not habituated to its use. It seems scarcely possible that the benefit in these cases comes from the stimulant effect alone. It is more likely that the alcohol acts directly upon the microbe by mingling with the medium in which it grows, or that it has an antidotal effect upon the poison already in the circulation.

Squire[79] reports a case of pneumonia occurring in hospital practice in which the patient became apparently moribund and was given up as hopeless. Brandy was given steadily by the interne and nurse until in twenty-four hours thirty-two ounces had been taken with decided benefit. By following up this treatment the patient recovered.

Abbott[80] reports a case of double pneumonia in a young woman of eighteen years, the lower half of both lungs being consolidated. All nourishment was refused, even milk, but she was induced to swallow brandy properly diluted, and was carefully watched for any evidence of overstimulation. Sixteen ounces of brandy were given her daily for three successive days. On the fourth day whiskey was substituted, and of this she was given twenty-four ounces daily four days in succession without evincing symptoms of discomfort or overstimulation. On the morning of the eighth day she was much improved and refused absolutely to take further stimulants. These were, therefore, discontinued, and she then took milk and other light nourishment without objection and made a prompt and complete recovery.

I have myself employed very large doses of alcohol in several cases and sometimes with marked benefit.

Digitalis in recent years has held a prominent place in the treatment of pneumonia. With the recognition that the chief danger is from heart failure, it is natural to expect benefit from the use of a drug that is of such great service in cardiac diseases in restoring the rhythm of the contractions, reducing their frequency, and giving them greater force and efficiency.

But the mechanical conditions in the two affections are as different as is possible to imagine, and the action of digitalis as an arterial constrictor, which in the one case is so beneficial, serves in the other case only to add to the labor of the organ which we are striving to sustain. Theoretically, then, digitalis scarcely seems to be indicated, and practically, while it is much employed, we have little or no evidence that it is useful as generally administered. Personally I do not resort to it unless there is cardiac arrhythmia, when I have sometimes found that the pulse became regular under its use. On the other hand, I am positive that I have often seen distinctly harmful results from its exhibition, at first in my own practice, and later in the practice of others. I have seen the evidences of dilatation of the right cavities become more marked, the lips more livid, and the chest râles more abundant; and I have seen these conditions subside when the digitalis was withdrawn, and its effects upon the peripheral circulation counteracted by the free use of nitroglycerin. Indeed, with my present views I consider the use of the drug in pneumonia without the simultaneous employment of an arterial dilator a highly dangerous and indefensible measure, and one that has contributed in no slight degree to the fatality of this disease.

Sturges and Coupland[3] remark: "For ourselves we desire to say that having used it frequently in pneumonia when in want of a cardiac stimulant where there was much dyspnœa, cyanosis, and other signs of auricular distention we have not found digitalis efficacious in removing those symptoms."

Larrabee[4] remarks that digitalis stimulates a weak heart by contracting the arteries and arterioles and throwing the blood back upon the heart itself. If there is no pulmonary obstruction the action is prompt and efficient, but the very condition which is killing the patient in pneumonia is rendered still more dangerous by such an agent.

Nevertheless, in the past ten years there has accumulated evidence to show that digitalis employed by a peculiar method may be of service in pneumonia. Beginning in 1888, a series of articles have been published by Petrescu, of Bucharest, in which he advocates the use of enormous doses of digitalis leaves, and claims for the method results that far surpass those of any other plan of treatment yet suggested. His daily dose reached the startling figure of 60–120 gr. of

the dried leaf. So far from any poisonous effects resulting he claims that in nearly every case a very prompt and decided amelioration of all the symptoms takes place, and that in six hundred cases the mortality has been less than two per cent.

Belotti and others have published articles confirming the result obtained by Petrescu. Such a remarkably successful treatment, and withal one so easily carried out, should, it would seem, have received universal favor. Admitting its claims to be well founded, we must seek a rational explanation first of the impunity with which such large doses are taken, and second of the beneficial effects obtained. As to the first point, it is to be observed that the preparation of digitalis employed is the infusion. Recent investigations show that of the four active principles obtained from digitalis, viz., digitalin, digitoxin, digitonin, and digitalein, the first is almost insoluble in water, the second quite so, while the other two are freely soluble in this menstruum. All are soluble in alcohol. Now in an infusion prepared without the addition of alcohol, such an infusion, for example, as that of the British Pharmacopœia (the United States Pharmacopœia prescribes ten per cent. of alcohol), there is a very small amount of the digitalin and no digitoxin, while the full amount of the digitalein and digitonin contained in the leaf is represented. Digitalin seems to possess to a remarkable degree the power of contracting the peripheral vessels, since Porter found that the capillary circulation in the web of a frog's foot was completely arrested by the application of a solution of 1:30,000. It is probably this principle, therefore, that gives to digitalis its power to raise the blood pressure. Its exclusion to a large extent from a purely watery infusion would therefore lessen the injurious effect that would otherwise result from the increased labor required of the heart to overcome increased peripheral resistance.

Digitoxin is supposed to be the most poisonous principle contained in the leaf. To its action is probably due the vomiting so often excited by the tincture or other preparations containing alcohol. Its beneficial action, if it possesses any, is not yet determined. It seems, therefore, that the remarkable effects obtained by Petrescu may fairly be attributed to the digitalein and the digitonin which the watery infusion contains. That this preparation will act to steady an irregular heart, and to give force and efficiency to the beat, as well as to increase the flow of urine is a matter of frequent observation. In a case of cardiac dilatation now under treatment by the writer the arrhythmia was excessive and the pulse extremely small, while the daily excretion of urine was only twenty ounces, yet under six daily doses of two drachms of the watery infusion the rhythm was

nearly restored, the pulse became larger and stronger, and the urine increased in two days to seventy ounces.

Statistics from military sources, like those of Petrescu, are open to this fallacy that the conditions, at least in time of peace, are peculiarly favorable for recovery. In Continental European armies the soldiers are all young men from eighteen to twenty-five years of age. Men recover in larger proportion than women to begin with, and younger men more frequently than the average subject. The soldier is not likely at his age to have damaged kidneys or an impaired heart as something acquired after entering the army, and before that time such a condition if present would prevent his enrollment. The same is true of syphilis and other cachexiæ. The subjects of the disease, therefore, are almost as if they were selected with reference to their ability to resist an attack of pneumonia. But this is not all. Garrison duty is irksome, and the young recruit is glad to escape from it on the slightest pretext of illness. Hence at the first symptom he reports to the surgeon, and the hospital is immediately at hand to receive him. This secures the advantage of early treatment and avoids the dangers of removal to a hospital after the disease is in progress. How potent the last two factors are may be judged from the statistics of the United States army, which being maintained by enlistment and reënlistment contains many men past the middle period of life and many alcoholics. Yet despite these unfavorable conditions and the hardships of frontier campaigning and Indian fighting, pneumonia is only about one-third as fatal in military as in civil practice.

Taking all those points into consideration, we should expect a very low rate of mortality from pneumonia in Continental armies in time of peace. Yet with allowance for all this, the results obtained by Petrescu are remarkably favorable, and it is singular that the special advantages of his treatment have not won for him a more enthusiastic support.

It is only fair to say, however, that other observers following his plan have not attained his success. Havas,[*2] for example, has given the method a trial and reports against it.

Application of Cold.—The high temperature attending many cases of pneumonia naturally suggests the use of cold applications either locally to the chest or to the surface generally. But the popular idea that pneumonia is usually if not always the result of catching cold, and the resulting prejudice against what would be considered a repetition of the exciting cause, have prevented its use to a great extent in private practice and even in hospitals. And it must be admitted that theoretically a long-continued impression of cold upon

the surface, contracting the peripheral vessels and forcing the blood into the interior, does not commend itself as a plan of treatment in a disease in which vitally important internal organs are already engorged. Nor does the result of practice favor such a use of cold with the idea chiefly of reducing the temperature. In fact, experience shows that when pneumonia occurs in the course of typhoid fever, the bathing, which before was not only well borne but was obviously beneficial, becomes a source of danger unless employed with the utmost caution. But these considerations do not apply to the local application of cold in proper cases, nor to its general application in such measure as to insure a prompt reaction with accompanying nervous stimulation and more active cutaneous circulation.

Already in 1888 Liebermeister upheld this method, and showed that when properly applied it not only was devoid of the dangers formerly attributed to direct refrigeration of the surface, but that the patient breathed more deeply, expectoration was freer, and a beneficial nervous stimulation resulted. The bath was usually given at 68° F. and was ordered when the temperature reached 104° F. The duration of the bath was about ten minutes. Cold sponging was resorted to between the baths if the temperature showed a tendency to rapid increase. He referred to one hundred and fifty cases treated by him in this way at Basle, with a mortality of ten and a half per cent. as against twenty-five per cent., his average under the older methods.

A case treated with cold sponging at the Presbyterian Hospital is illustrated by the chart on opposite page.

Different observers, and especially Mays, have found marked benefit from the use of ice bags or ice poultices to the affected side of the chest. The effect of these applications cannot be directly to lower the temperature of the diseased structure. Gilman Thompson has shown that ice applied to the surface of the abdomen does not sensibly affect the temperature of the corresponding interior surface of the abdominal wall. Much less could ice in contact with the chest, although affecting to some degree the general temperature, be expected to abstract an appreciable amount of heat from the underlying lung. But this does not preclude a reflex influence that may affect powerfully the vascular action in the organ involved, as it is well known that the supply of blood to a viscus may be controlled to a marked extent by impressions produced within areas nervously related to it. In any case, there is abundant clinical testimony to the value of cold applied in this manner. The results of a collective investigation instituted by Mays,[*3] and covering one hundred and ninety-

Admitted on 8½ day
Crisis on 12th day
Treatment strictly
by sponge baths
Chart No. 7.

five cases, however, had to do with private practice, where the mortality is relatively small. The figures are not therefore so favorable as they appear to be at first sight.

The method consists simply in packing bags, filled with chopped ice and covered with towels, about the affected side of the chest. The pain is relieved, the breathing becomes deeper and less frequent, the temperature and pulse decline, delirium ceases, and a general improvement begins, which in most cases is permanent.

The general application of cold, however, has its modern advocates. Baruch,[54] following Liebermeister, advises the cold bath or the cold pack, but so administered as to arouse the nervous system and stimulate the cutaneous circulation rather than with a view to direct reduction of temperature. The bath, therefore, is of short duration, and is accompanied by brisk friction, or if the pack is used, blankets are placed outside, so that the impression of cold is quickly followed by reaction and perspiration.

Peabody[55] considers the use of the tub bath precisely as in typhoid fever as marking the most important advance in the treatment of pneumonia for many years. My own experience in this line is limited to the use of the wet sheet about the trunk. In a number of cases this has seemed to be serviceable.

Antipyretics.—From what has gone before, it will be seen that there is not much room for antipyretics simply as such. The cases are rare in which benefit can be expected from merely forcing down the temperature by the use, for example, of the coal-tar preparations. Yet when with hyperpyrexia there are great discomfort and much nervous disturbance, such as headache, jactitation, insomnia, delirium, etc., and there is a fairly good condition of the pulse, one of these agents may be cautiously employed, its use being suspended so soon as a measure of relief has been obtained. The main thing is not to follow the indications of the thermometer alone, but those afforded by the general symptoms plus the thermometer.

The high temperature is not of sufficient duration to threaten of itself the integrity of the heart fibre, as it does in the case of a continued fever. The risk is from the local action of the toxin, and we add to this risk when we resort to antipyretic drugs. We are thus confined to a discriminating choice of evils.

As to which of the coal-tar products is to be preferred, while phenacetin is much in use, my personal choice is acetanilid. A combination of three grains of this and a like quantity of Tully's powder has given me much satisfaction. The small amount of morphine intensifies the soothing effect, and with the camphor acts as a stimulant to the heart, and at the same time tends to promote the action of the skin.

Relief of Pain.—When the pain is severe, nothing else will take the place of a properly apportioned dose of morphine given hypodermatically. It not only relieves the suffering, but it promotes deeper, slower, and more efficient respiration, and thus lessens the tendency to capillary stasis in the affected portion of the lung. But large doses are to be avoided, and it should be used very cautiously when there is any considerable loss of respiratory surface or any tendency to somnolence. A quarter or a third of a grain will usually be enough, and the dose may be cautiously repeated. In a doubtful case it may be more safely used in combination with a full dose of caffeine.

If in any case the use of morphine is deemed inadvisable, the pain may be alleviated by the application of an ice bag or ice poultice. In sthenic cases wet-cupping, or the application of a dozen leeches, may accomplish the immediate purpose, and at the same time exert a favorable influence on the progress of the disease. When the pain is less severe the application of dry-cups, sinapisms, etc., may suffice. Poultices, formerly so much used, are now less in favor. They are mussy, troublesome to apply, apt to become cold and clammy, and unless in the hands of a very faithful and assiduous nurse, are likely to do more harm than good. Even the time-honored oiled-silk jacket is now seldom seen, and will doubtless soon become obsolete. The jacket, made by quilting cotton batting between thicknesses of cheese-cloth, has the negative merit of being comfortable and cleanly, and is at least unobjectionable.

When the pain is low down, a single broad strip of adhesive plaster, drawn tightly around the affected part, allowing the ends to extend an inch or two beyond the meridian line, will give very marked relief. Higher up, where the movement of the ribs is less, this expedient is less useful, but it is still not entirely without benefit.

Relief of Cough.—In some cases the cough is out of proportion to the material to be expectorated, and exhausts the patient with no corresponding benefit. Worse than this, convulsive coughing tends to aspiration of the sputum into the unaffected portions of the lung, thus increasing the area of infection.

For both these reasons it is important that an irritative cough should be held in check. This may be accomplished by the moist inhalations already mentioned; but if these are not sufficient, small doses of codeine or morphine will be required. The new drug, heroin, which is a substitution-product of morphine, seems to have a special power to control cough, while not producing in the same degree the other effects of the parent drug. In doses of about gr. $\frac{1}{12}$ it has

given me great satisfaction, a single dose sometimes moderating the cough for several hours.

Œdema of the Lungs.—The supervention of pulmonary œdema during the progress of pneumonia is a very grave occurrence. It is indicated by abundant coarse râles over the whole chest, very rapid and superficial breathing, frequent and small pulse, more or less cyanosis, and profuse and often cold perspiration. It may appear with comparative suddenness, and be very promptly fatal, the patient dying of suffocation.

The treatment must be prompt and vigorous. The chest should be covered with dry-cups, or enveloped in a huge mustard poultice. A small hypodermic injection of morphine, not more than gr. $\frac{1}{8}$, will do more than anything else to restore the pulmonary circulation and check the excessive secretion, which sometimes wells in white or pinkish foam from the mouth and nostrils. In the same injection should be included gr. $\frac{1}{75}$ of nitroglycerin and gr. $\frac{1}{60}$ of strychnine. Oxygen if obtainable should be given freely by inhalation. Half a pint of very strong hot coffee should be thrown into the rectum. By these means some apparently desperate cases may be carrried through successfully. As a last resort, artificial respiration may be employed to aid in pumping the air and oxygen into the lungs.

Résumé.

In the view of the writer, the treatment of pneumonia should embrace the following points:

An attack upon the pneumococcus through the medium of the blood, the object being that the exudate when it escapes into the air cell shall be impregnated with a substance that will unfit it to serve as a culture medium.

Stimulation of the emunctories to throw off the poison as it forms.

Sustaining the vital powers and particularly the heart—cardiac stimulants.

Relieving the pulmonary circulation—vasodilators, venesection.

Compensation for loss of respiratory surface—inhalations of oxygen.

Reduction of excessive temperature—cold to surface, antipyretics (?).

Relief of incidental symptoms.

ANTITOXIN TREATMENT.

In all probability the pneumonic process as we see it from the moment of invasion to the crisis implies infection by the agency of a continually changing set of microbes. Welch has shown that the

virulence of the pneumococcus is inversely as its age, the organisms taken from the centre of a pneumonic focus having very little potency, while on the edge of a patch which is still spreading they are most active. From this we infer, and the inference is supported by the behavior of artificial cultures, that the infection of the system is not maintained through the whole period of pyrexia by the same microbes, but by a constant succession, the older ones becoming inert, and fresh once carrying on the work of supplying the toxin. This supply fails soon after the local process has ceased to spread, simply because there are no longer any young bacilli to maintain it. But while it would seem as if this might afford a sufficient explanation of the phenomena of defervescence by crisis, yet the observations of the Klemperers and others make it reasonably certain that there is an antitoxin produced that has its share in the result. Whether this antitoxin is the product of the pneumococci or is simply the result of changes going on in the leucocytes preparatory to their disintegration is not yet determined. We are apt to think of the process of absorption by which resolution is ultimately effected as beginning *after* the crisis, whereas it is in operation from the first moment of the disease, as is shown by the early infection of the general system which it brings about. Up to the point of consolidation the rate of deposition is far greater than that of removal, and a rapid accumulation of exudate takes place in the air cells. This exudate, however, is not permanent in constitution. It immediately begins a process of change, and as we have seen, the organisms contained in it change their properties also. As time goes on, there comes a period when the deposition of fresh infective material is less active than the absorption of that which is older. At this point an antitoxic effect becomes apparent, and in the cases terminating by crisis, a rapid fall of temperature takes place. In the cases terminating by lysis, either the deposition of fresh material is protracted by the invasion of new territory, or the absorption of the older material is for some reason less active.

As early as 1888, Netter rendered mice and rabbits immune to pneumonia by injecting them with fluid prepared from the dried spleen of infected animals. Later he experimented with an old pleuritic exudate containing pneumococci, and at last the sputum of a pneumonic patient, which had ceased to be virulent after the crisis.[86]

Pursuing this line of investigation, Foà found that the injection of an attenuated culture of the diplococcus of pneumonia into an animal gave immunity against the disease for several months. He produced the injection serum by precipitating the culture broth con-

taining the diplococci with ammonium sulphate and filtering repeatedly. The filtrate was injected into the veins of rabbits daily for three or four days. Subsequently he made an extract of the muscles and viscera of a rabbit which had died from pneumonia, precipitated it in the same way, and found that it gave the same immunity. The extract from a healthy rabbit when injected gave no immunity.

He next turned his attention to the immune animal, drawing the blood and allowing it to coagulate, when the serum was used to inject another animal. This also became immune. As a control experiment, he injected a rabbit with blood from a man dead of pneumonia; death resulted. He states that several species of virus are formed, one seeming to act on the nervous system, another on the blood and tissues.

The Klemperer brothers verified these results.[77] Their report is briefly as follows: Two rabbits were injected each with 20 c.c. of pleuritic exudate taken from a pneumonia patient and which by culture was shown to be free from living bacteria. Fourteen days later both were inoculated with a virulent culture. Both survived, while the control animal died. Later, they immunized animals with pneumonia sputum taken before the crisis and heated so as to destroy the poison. The same result was obtained by heating to 60° C. a glycerin extract of pneumococci. The bacteria were washed from agar cultures with sterilized glycerin, which was exposed to heat for one or two hours and filtered repeatedly. They found that immunity resulted from doses proportioned in quantity to the strength of the preparation. It was employed subcutaneously.

They found further that dogs can be immunized against pneumonia, and can also be cured of the disease. This cure takes place through the serum of immune animals, immunized by taking in the products of the activity of the pneumococcus. This immunizing serum does not cure by killing the cocci in the system, for after four days' contact with the serum the bacteria injected into an animal caused death. In fact, the bacteria in contact with this serum increase in number. At the same time the serum if injected prevents the formation of the poison in the body of the animal. This may be explained in one of two ways, either the serum hinders their power of forming poison, or the cocci go on forming poison, and the serum counteracts its effects, or through chemical changes renders it inert. At any rate, the cocci become harmless to the animal, their evil influence being destroyed by the reaction of the body cells, especially the white blood corpuscles.

If pneumotoxin and curative serum be mixed and injected into an animal, there is no rise of temperature and no effect from the

poison, while toxin alone kills the animal, with symptoms of septicæmia.*

They next inquire whether the pneumonia cured in animals by the serum is identical with pneumonia in man. Their autopsies in animals dead of pneumotoxin injections did not show the fibrinous exudation in the lungs characteristic of pneumonia, but diplococci were found in the blood. However, it is not the pneumococci themselves, but the poison that they produce that gives the septicæmic symptoms. This poison when formed at the seat of injection reaches the blood sooner than the cocci themselves. Also if we filter out the cocci, the poison remaining in solution kills the animal injected as effectually as the original culture.

In contradistinction to animals, man is only slightly susceptible to the pneumococcus. Living cocci can exist on healthy mucous membranes without doing harm, and are found in the sputum of healthy men. If we inject men subcutaneously, what results do we get? The Klemperers experimented by injecting themselves, and found that no reaction resulted from small amounts; from larger ones a local swelling with rise of temperature and febrile symptoms resulted, passing away in a few days. Hence, they conclude that men are much less susceptible than dogs to the same relative doses.

They conclude that in man it is not the exudation in the lung that renders the disease so grave, but the general infection from absorption into the circulation acting on the heart and vital centres and producing febrile symptoms. In the animals injected, the poison enters the blood stream directly and produces fever at once. The poison increases for some days, and then antitoxin is produced and immunity results. Serum taken from pneumonic patients after the

* In this connection may be mentioned the observations of Fochier of Lyons, who recommended the production of abscesses by subcutaneous injections of turpentine in certain cases of puerperal infection. This recommendation was carried out in pneumonia by Lépine and others with gratifying results. Pinna[28] in observations already referred to (see page 44) shows that pure sterile pus has an antitoxic effect in pneumonia, and that this may be made available by Fochier's method, viz., the injection of 1 c.c. of essence of turpentine beneath the skin, thus inducing the formation of a sterile abscess. He treated in this way a patient, fifty-five years of age, suffering from apyretic and adynamic pneumonia of seventeen days' standing, in whom the diagnosis was based upon physical signs and the presence of pneumococci in the expectoration. The day following the injection the pulse rose. The temperature went suddenly up to 102.2° and the dyspnœa was relieved. Within a few days the temperature fell again to normal, and the pulmonary phenomena subsided. Meantime an abscess had formed at the site of the injection which when opened gave issue to 50 c.c. of perfectly sterile pus.

While this practice is not likely to find many imitators, the observation is of value in the discussion of the phenomena of antitoxin production.

crisis is found to cure pneumonia in dogs. They conclude that from pneumotoxin in man is produced antitoxin at the time of the crisis, and this counteracts the effects of the toxins. Thus the so-called crisis in man is the beginning of the formation of the antitoxin, and though the cocci remain for some time in the blood they are no longer harmful.*

They then question whether immunity in man results after the crisis, and conclude from researches and experiments that it does occur, but is only temporary.

With regard to the treatment of pneumonia, they say: At present we use supportive measures, awaiting the formation of antitoxin. But the aged and weak succumb. They suggest that by using the immunizing serum from animals, we may hasten this process and save lives. They have treated some cases in this way with apparent benefit, as shown by fall of temperature and slowing of pulse and respiration. But not enough have as yet been treated to arrive at a definite and final conclusion as to the value of the treatment.†

An important discussion on this subject took place at the Academy of Medicine in Turin, December 2d, 1892.[86]

Lara, chief physician of the Hospital San Giovanni, reported the results of ten cases of pneumonia under serum treatment. Five of these cases were double, five single. Eight of the patients were young persons, two advanced in years; six were robust, four of debilitated habit. The serum was in some cases from immunized rabbits, in other cases from dogs, and in still other cases a glycerin extract made from the viscera of refractory animals was employed. In no case was there any local reaction. The glycerin extract produced no observable general symptoms. Serum from dogs caused nervous excitement; that from rabbits produced general agitation and a temporary aggravation of the disease. In all but three cases there was reduction of temperature, not sudden, but after an interval.

A change in the character of the pulse was observed without a reduction in the number of beats. There was no immediate change in the respiration, but after a time it became somewhat slower. The crisis took place in from three to five days. The convalescence was rapid and complete; complications were rare and of little gravity. The reporter considered the results encouraging.

* This statement is not in accord with more recent observations which show that cases in which cocci are found in the blood generally prove fatal.

† Pana and di Renzi, of Naples, have produced a serum by inoculating animals with pneumonia bacilli, and have employed it in some 32 cases with 29 recoveries and 3 deaths. In respect to all of the latter the autopsy showed the presence of other diseases of a fatal nature. Maragliano experimented with this serum in five cases with success in all (Antonio Fanoni[87a]).

Bozzoli reported five cases treated with serum from rabbits prepared by a special process not described. There was rapid fall of temperature in every instance. Four of the patients recovered and one died after defervescence. The kidneys were unaffected.

Di Renzi reported that during the past year he had treated ten cases of pneumonia with antipneumococcic serum, prepared as follows: The animals were inoculated with a non-lethal quantity of pneumonia virus, the dose of which was gradually increased until a strong immunity was produced. Serum from these animals was injected into the patient. Only severe cases were selected for treatment. In every case cure resulted. In one case the temperature came down on the third day, although there were signs of diffuse hepatization. Of five other cases admitted during the year, and not treated with serum, one died. Although the author admits that his cases might have recovered without the serum treatment, he considers his results decidedly encouraging, as pointing to a real and efficient treatment of pneumonia.[89]

Wiesbecker[90] reports five cases of pneumonia treated with injections of serum obtained from patients recovering from the disease. While there was no uniformity in the results so far as the objective signs were concerned, these becoming more and more severe in some cases after the injection, while in others the severity abated, there was in every instance a most remarkable improvement in the subjective conditions. This improvement was almost instantaneous, in one case being manifest within one and one-half minutes after the injection. But for one instance in which the patient was a child only three years of age, we should be inclined to refer these marvellous results to suggestion, especially as they did not conform to the physical conditions present at the moment. Difficulty of breathing, pleuritic pain, malaise of every description disappeared as if by magic; recovery took place in every case, though not always with remarkable promptness. The quantity of serum in each case was 10 c.c.

Similar to this was the experience of Fourriere[91] in a single case treated by injections of goat's blood. The patient, a person past middle age, was recalled to life from an unconscious, fairly moribund condition, and though death occurred after several days, there was an interval during which danger seemed to be over and recovery assured.

In 1897, Washbourn, of Guy's Hospital, published his researches on antipneumococcic serum. His method was essentially that employed in producing diphtheria antitoxin. A pony was the animal selected, and after nine months' treatment, first with living and then

with dead cultivations, the serum was found to possess marked protective powers. By using a special method of cultivation, it was found possible to maintain the virulence of the pneumococcus at a given level for a period of sixty-six days. To maintain the virulence of this culture it must be kept in an incubator at a temperature of 37.5° C. He claims that under these conditions the antipneumotoxin can be accurately standardized.[92]

Several cases of pneumonia have been treated by Washbourn and others with this serum. While its influence cannot be distinctly traced, it can be fairly considered to have contributed to the favorable result in some extremely unpromising cases.

We cannot avoid the conclusion from this résumé of the achievements of orrhotherapy in its application of pneumonia, that up to the present time they can scarcely be said to amount to more than an encouragement to further effort. No really decisive results have been obtained. In some cases the effect seems to have been favorable, but in view of the variable course of pneumonia under all forms of treatment, it is impossible to assign to the injections any positive share in the result. It can be only by the accumulation of a large number of observations that a conclusion as to the value of the treatment can be arrived at, and, unfortunately, the difficulties in the way of extended observation are such as to deter most investigators from pursuing the subject.

The first difficulty is found in the short life of the pneumococcus and its feeble power of resistance. Cocci that are virulent at the beginning of an investigation cease to be so as the investigation proceeds. On the other hand, toxins that are expected to produce only a moderate reaction when injected sometimes display an unlooked-for virulence. Animals apparently progressing normally towards immunity most unexpectedly succumb to septicæmia from a dose of toxin supposed to be entirely within the limits of safety. Again, animals that were readily immunized at first lose their immunity in spite of renewed inoculations, and the serum obtained from them ceases to be reliable. This variation in the conditions under which experimentation is conducted is liable to vitiate the most carefully drawn conclusions. If this be true under the favorable circumstances of the laboratory, what must it be in the exigencies of ordinary practice? If before employing a therapeutic agent we must resort each time to experiment to test the value of the specimen in hand, the usefulness of the agent will be very limited.

It is to be hoped that this difficulty has been overcome by Washbourn's method already mentioned, and that it will be possible in the future to command a supply of reliable antitoxin for the treatment of

pneumonia, as we already do for the treatment of diphtheria. Efforts to produce such a supply are now being made by the Health Board of New York. Should they be successful, the value of the method will soon be determined.

Both Bonardi and Griffiths succeeded in isolating crystallizable substances which were supposed to represent the active principle of the pneumotoxin, the first from cultures in artificial media, and the second from the urine of pneumonic patients. But their conclusions seem to have been negatived by subsequent investigations carried out at the Institute for Experimental Hygiene at Rome, by Alfredo Andreini,[98] so that we are still ignorant of the chemical properties of the poison, and as far as ever from the brilliant therapeutic results which it was thought were about to be realized.

Pneumonia in Childhood.

Pneumonia in childhood differs in some respects from the disease as occurring in adults.

As young children do not expectorate, the study of pneumonia in them is deprived of the aid to be derived from the examination of the sputum, so useful in the case of older persons. As most cases end in recovery, the opportunity for autopsical research is limited. For these reasons our knowledge of pneumonia as it occurs in childhood is relatively incomplete.

In the Kaiser und Kaiserin Friedrich Kinderkrankenhaus at Berlin, Schlesinger found that in children up to fourteen years of age genuine fibrinous pneumonia occurred in the proportion of thirty-seven to two hundred and seventy cases of bronchopneumonia. Holt[99] states that after three years of age nearly all primary cases of pneumonia are of the croupous variety. During the first two years twenty-five per cent. of the cases are croupous and seventy-five per cent. bronchial.

Croupous pneumonia appears in earliest childhood, it has been even recognized at birth. It attains its greatest frequency in the fourth year, after which the morbidity decreases until puberty. From earliest infancy, when the mode of life is the same in both sexes, boys are more frequently attacked than girls, in the proportion of eleven to seven (Schlesinger,[4] Morrill[5]). Children of vigorous constitution are not more exempt from attack than the less robust. Zymotic disease and acute gastritis play an important part as exciting causes. The season of the year has the same influence as in adults.

Symptoms.—In about half the cases the attack begins with vomiting; in about seven per cent. with convulsions (Holt).

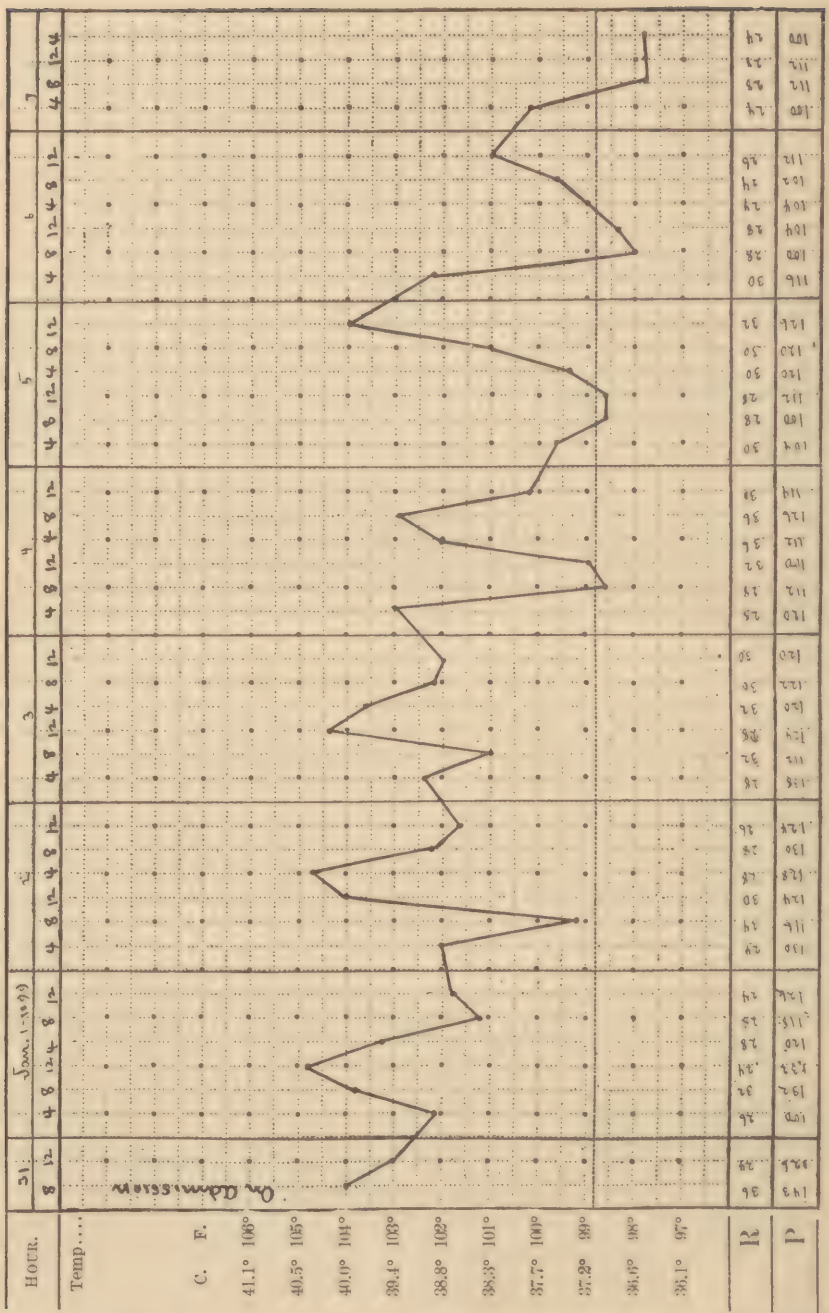

Chart No. 8.—Showing the Intermittent Temperature Quite Common in Children.

As a rule, the younger the child the higher the *temperature* range, and the more frequently the fever assumes an intermittent or remit-

tent form, contrasting in this with the comparatively even temperature observed in the adult. In pneumonia of the upper lobe the fever generally is higher and more constant than when the lower lobe is the seat of the disease; but there are exceptions to this rule, as in the case of which a chart (No. 8) is here presented. Morrill[95] found the highest temperature in seventy-two cases to be 106.5° F., lowest maximum 101° F. Average critical day in fifty-four cases, the eighth.

As compared with adults defervescence by lysis occurs less frequently in children, in proportion as the pneumonia is purely lobar and not bronchial, and the fall of temperature at the crisis is more marked. Especially in very young children, and in disease of the upper lobe subnormal temperature after the crisis is the rule, sometimes to the extent of 2° or 3° F.

Procrisis as distinguished from pseudocrisis appears to be a peculiarity of childhood.

While the disease is at its highest the temperature, pulse, and respiration remain nearly parallel with each other. At the crisis, the first two fall abruptly, while the respiration sinks more gradually. After the crisis the relative frequency of pulse to respiration not uncommonly falls below 2:1. This, in children, is very rarely the case before defervescence. Excessive rise, the pulse, for instance, above 170 and the respiration above 75, is rare in croupous pneumonia, as is also a slowing of the pulse to below 80 after the crisis is past.

The lesion of croupous pneumonia does not observe the boundaries of the lobes nearly so accurately in children as in adults. Much more frequently the invasion of a neighboring lobe remains only partial. Dulness on percussion is more marked than the auscultatory signs, and will often be apparent in cases that without it would be overlooked. It is never safe to exclude pneumonia in a child simply because we fail to get bronchial breathing and bronchial voice. The percussion should be light and with a single finger.

Holt observes that when the appearance of physical signs is delayed, we should look for them in the axillæ and in the mid-clavicular regions in front.

Double pneumonia is less frequent in children than in older persons, while an attack limited to a single upper lobe is relatively common. When several lobes are invaded it is usually in succession, and there is an ascending scale in the severity of the attack. In twenty cases observed in the New York Foundling Hospital* the

* Private note from Dr. Kauenhoven.

right lower lobe was primarily involved in seven, the left lower lobe in eight, the right upper lobe in three, the left upper lobe in two.

The age of the youngest child with distinct signs of lobar consolidation was five months. There were three cases in children six months old and under. In all cases with only one lung involved defervescence took place on or before the sixth day. All uncomplicated cases terminated in recovery. Of the two fatal cases one was complicated with rachitis and one with croup. Both were in children under one year of age.

Morrill[96] gives the location of the lesion as follows: right middle or lower lobe, 40 per cent.; left lower, 25 per cent.; right upper, 10 per cent.; left upper, 8 per cent.; right apex, 5 per cent.; left apex, 4 per cent.; both lungs, 5 per cent. Mortality, 5 per cent.

Resolution progresses more rapidly than in average cases in the adult. Unresolved pneumonia is rare, and the issue in gangrene or in phthisis is almost never encountered.

The normal course of pneumonia in childhood is more severe than in adult life. Right-sided cases are more severe than those on the left, but pneumonia of the upper lobes is not more serious than that of the lower. After a prodromal stage of one or two days which in children is not infrequent, the disease develops without marked initial symptoms. These, however, are seldom wanting when the attack is sudden and without prodromes. A pronounced chill is much less frequently observed than in grown persons (Morrill found it present in only five per cent. of one hundred cases), while vomiting is very common. Convulsions are rare and occur for the most part only in very young children, and in right-sided attacks. Just before the crisis the child often is much sicker than at any previous stage. The contrast then with the subsequent rapid improvement is most striking.

Still in a small fraction of cases the recovery does not begin at once with the fall of temperature, but a day or two days of exhaustion and depression intervene.

Abortive Pneumonia.—Baginsky[97] describes under this title a form in which the attack sets in suddenly, giving rise to the suspicion of croupous pneumonia by the type of the respiration, the temperature, and the pulse, and yet does not come to the full development of the physical phenomena. Rather, the process seems to be arrested halfway; before it comes to actual consolidation the fever breaks, and the morbid conditions disappear as rapidly as they arise. He considers these as undoubtedly cases of pneumonia that have retrogressed in the stage of engorgement which always precedes consolidation. In his practice such cases are not very infrequent.

Under the title of "wandering pneumonia," he observes that

while it is not very common that pneumonia spreads from lobe to lobe or invades in succession the entire lung, yet such cases do occur. By physical examination it is possible to follow these several developments. The cases divide according to the temperature curve into those in which the fever remains constant, and those in which there is a decline of temperature followed by another rise. While we have to do in the first case with a single protracted attack, the other represents the recurrent type of pneumonia in which a new section of lung is invaded. All attacks of this kind have a long duration and follow a severe course.

Baginsky also recognizes a type which he terms "gastric pneumonia." In this form the gastrointestinal phenomena are in the foreground, while the affection of the lung, until shortly before the critical appearance, assumes but little prominence. These cases are apt to begin with vomiting, and diarrhœa is a leading feature. Only when the disease has made considerable progress does the pulmonary implication appear in its true character as the essential element in the case.

Among the anomalies in the progress of the disease, an abortive action is observed more frequently in distinct but circumscribed infiltration than when only the stage of congestion is reached. The most dangerous anomaly in children is the migratory tendency. The difference in the varieties it occasions lies in the duration of the intervals between the several migrations. A recurrence of pneumonia is as rare in children as in adults, though relapses may occur and the attack is greatly prolonged in consequence. Stockton[7] reports a case in which the entire duration was sixty-eight days.

The most frequent of the abnormal developments is "cerebral pneumonia." A convulsive, a comatose, and a delirious form are distinguished. The cause of the brain symptoms may be found in the pyrexia, in the severity of the infection, in an otitis media, or finally in an individual peculiarity of the child. A genuine meningitis as a complication of pneumonia is apt to follow an almost latent course.

The examination of the blood shows that the leucocytosis follows essentially the same course in the pneumonia of children and of adults.

Complications.—A slight bronchitis is often observed in the beginning of the attack in a child, being apparently a continuation from the prodromal stage. Later it is of rare occurrence, but of more serious moment. Pleurisy is the most frequent complication, especially in pneumonia of a lower lobe. It is even more frequent than in the adult. For the most part it follows a favorable course, seldom pass-

ing into empyema. It influences the fever but little, but even when it is slight, it delays more or less a complete recovery. Albuminuria is remarkable for the relative rarity of its occurrence in the pneumonia of children. When present it is slight in amount and of short duration.

Herpes is relatively uncommon. Icterus is lighter and less frequent in childhood than in later years.

Otitis media is especially a complication belonging to childhood. Its course is generally severe, and often accompanied by cerebral symptoms. It aggravates the fever very materially. As regards the ear itself the inflammation usually runs a favorable course. Otitis occurs for the most part during the first three years of life and in cases of pneumonia affecting the right side.

Prognosis.—The mortality of pneumonia in children is markedly less than in adults, in round numbers about one-third as great. Morrill gives the mortality in seventy-two cases as 1.5 per cent. The fatal cases are largely the result of complications of which the most important are meningitis, purulent pericarditis, and bronchopneumonia. As affecting the prognosis, the constitution and, in a less degree, the age, are to be taken into account.

As to the *diagnosis*, many of the peculiarities in the course of pneumonia in childhood spring from its relation to bronchopneumonia, which has yet to be sharply differentiated from it. The secondary pneumonias are very seldom croupous.

In the *treatment* a constant progress towards greater simplicity is the modern tendency. Energetic measures are less frequently resorted to. For lowering the temperature cold packs are coming into favor as against the use of the coal-tar preparations and other internal antipyretics. While the latter depress the already weakened heart, the former, if judiciously applied, improve its innervation and arouse its flagging energy. It is, moreover, to be recognized that up to a certain point the increased temperature is a useful factor in the struggle of the organism against the specific infection, and that if it is not excessive its forcible reduction is to be deprecated. A proper use of tonics and stimulants, including alcohol, will be of service.

Bronchopneumonia.

Lobar pneumonia in children is ever prone to take on more or less of the characteristics of bronchopneumonia, that is to say, it is apt to appear in different localities and to be accompanied by signs and symptoms of bronchitis. The distinction between the two forms is not always sharp, and both may be present at the same time.

This subject has been studied recently by Samuel West.[39] The following is a résumé of his paper:

The author believes that under the one term "bronchopneumonia" several different conditions are included, and that many of them are pneumococcus inflammations; in other words, that some of the bronchopneumonias of children are really the same disease as the common pneumonia of the adult, or to put the proposition in another form, that the only difference between the pneumococcus disease of the adult and that of the child is that in the one case the consolidation which results is lobar or massive, and in the other lobular or patchy.

Speaking generally, bronchopneumonia is shown to be associated with several varieties of pathogenic organisms; for example, the streptococcus, staphylococcus, Friedländer's bacillus, the tubercle bacillus, and others; but chief among them all is the pneumococcus, which, taking all cases together, is present either alone or in association with some of the others named, in at least fifty per cent. Thus Netter examined forty-two cases, and found:

		Per cent.	
Pneumococcus alone in	10	19 = 45	
With others in	9		
Streptococcus alone in	8	23 = 60	
With others in	15		
Staphylococcus alone in	5	13	45
With others in	8		
Friedländer's bacillus alone in	2	6	
With others in	4		

Weichselbaum, in 15 cases, found the pneumococcus in 7, streptococcus in 6, Friedländer's bacillus in 1, and streptococci and staphylococci in 1. Horton Smith, in 11 cases, found the pneumococcus alone in 5, with others in 3—that is, in 8 out of 11, and in only 3 cases was it absent. Mosny examined 4 cases of primary bronchopneumonia, and found the pneumococcus in 3 and the staphylococcus in 1. In 13 secondary cases the pneumococcus was present alone in 1, and associated with the streptococcus in 1. The streptococcus was found alone in 5, and associated with other bacilli in 5 more. Friedländer's bacillus was present in 1, and an unknown bacillus also in 1.

So far as the evidence goes it seems to show that in secondary bronchopneumonia the streptococcus is the most frequent organism, and in primary bronchopneumonia the pneumococcus, and that among the latter the pneumococcus is almost as common as it is in lobar pneumonia of adults.

It is now well known that the microscopical distinctions often

drawn between the minute lesions of lobar and lobular pneumonia cannot be regarded as absolute, and that for example even the fibrin network which is so common in lobar pneumonia is not uncommon in acute bronchopneumonia, while it may be absent in lobar pneumonia.

The conclusions to which this evidence points are obviously these:

1. That the primary and secondary bronchopneumonias have a different bacteriological origin.

2. That secondary bronchopneumonia is for the most part due to streptococcus infection, derived from some source in connection with the air tubes, throat, or mouth.

3. That primary bronchopneumonia is of pneumococcal origin.

4. That pneumococcus inflammation occurs with almost equal frequency in the child and in the adult.

5. That pneumococcal inflammation takes a different form in each, in the adult producing massive consolidation and in the child disseminated patches of consolidation; in other words, that there are no real pathogenic distinctions between lobar pneumonia of the adult and primary lobular pneumonia of the child.

To sum up, the author is of the opinion that the term "bronchopneumonia" would be best reserved for those inflammations of the lungs which follow antecedent affections of the bronchi, and that their exciting cause for the most part will be found to be other organisms than the pneumococcus, while, on the other hand, the primary bronchopneumonia of children is really croupous pneumonia occurring in a disseminated and patchy form instead of a massive consolidation.

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